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# FOOD POISONING

## ITS NATURE, HISTORY AND CAUSATION MEASURES FOR ITS PREVENTION AND CONTROL

BY

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IN THREE PARTS
WITH APPENDIXES

*ILLUSTRATED*



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# FOREWORD

It is only during the past two or three decades that the real nature of the illnesses commonly grouped together under the popular term ‘ food poisoning ’ has been shown.

Modern research has proved these conditions to be due to the infection of food by definite organisms or their toxins. The food itself has little or nothing to do with the illness, except to act as a carrier or vehicle of distribution, just as water may carry the cause of typhoid fever or milk that of scarlet fever.

The work on this subject has been done by many observers in many places, and the results are recorded in many scientific papers and reports not readily available to the general, or even professional, reader.

The author of this work has done a great service in bringing all this work together in due perspective in one volume in a manner which would not have been possible a few years ago.

Here is a complete account of the whole subject which ought to dispel many erroneous ideas still prevailing and provide a reference for all those interested in this fascinating aspect of the public health.

GERALD LEIGHTON, M.D.

# AUTHOR'S PREFACE

In compiling this work, my intention has been to collect and present in readable form in one volume the fundamental facts relative to the many kinds of human food poisoning. The selection of essential material has been somewhat difficult, because a very large part of the information on the various subjects, especially bacterial food poisoning, is so widely distributed in numerous medical works, scientific treatises, journals and pamphlets, or recorded in Public Health Reports published during the past decade as a result of the investigations, studies and experiments by medical experts and observers in this country, the Colonies and the Public Health services and Universities in the United States of America.

No originality is claimed for this book. Many well-known works of reference have been consulted, and I gratefully acknowledge my indebtedness to the authors concerned.

Some interesting historical matter concerning early food-poisoning investigations has been included to indicate the sequence of events leading up to important bacteriological discoveries.

References are appended to each chapter for the use of readers desirous of consulting the original articles or books.

Quotations and Figures 28, 29, 30, 32 and 33 from official publications are included by permission of the Controller of His Majesty's Stationery Office, the Ministry of Agriculture and Fisheries and the Ministry of Health.

An Appendix on the Contamination (and Decontamination) of Foods by Poisonous Gases used in War has been kindly contributed by Mr. Henry Eastwood, M.R.San.I., Food Contamination Officer, Borough of Hornsey, London.

I am greatly indebted to Sir William Savage, M.D., for his valuable assistance, and my sincere thanks are accorded to Professor W. W. C. Topley and Professor G. S. Wilson for kindly permitting me to quote from their work on " The Principles of Bacteriology and Immunity " ; to friends, both at home and abroad, including Professor K. F. Meyer, Dr. J. G. Geiger, Dr. F. W. Tanner and Dr. S. R. Damon, for allowing me to make extracts from their writings, and to the Rockefeller Institute for Medical Research, New York, for consenting to excerpts being reprinted from their Monograph on " Botulism " by the late Dr. Ernest Dickson.

I am grateful, too, to all those who loaned photographs of some of the early investigators and to the publishers of " Food Manufacture," for their guidance and help. Finally, I must acknowledge the valuable help received from my wife in the preparation of the manuscript and index.

E. B. D.

Epsom 1943

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# PART I

## CHAPTER I

## INTRODUCTION

THE term 'Food Poisoning,' used in its broadest sense, embraces a variety of human ailments caused by poisonous substances transmitted by the food or drink ingested. In its strictly technical sense, however, it is confined to infections and intoxications associated with certain pathogenic organisms; the majority of outbreaks to-day are of this type.

This book is devoted to the following categories of food poisoning:

Bacterial food poisoning (including botulism),
Contamination of food by metals,
Plants and fungi,
Fish and shell-fish,
Contamination of food by poisonous gases used in war,

and includes food sensitisation or food allergy.

Bacterial food-poisoning outbreaks frequently occur during the summer months. Sometimes they assume large proportions, especially when the milk supply is the source of infection. Owing to a considerable number of outbreaks being of a mild and temporary type and limited to one or more persons or members of the family, they are frequently overlooked or are not investigated. Only when the malady is of a really serious nature and medical advice is sought, or when a considerable number of persons are attacked simultaneously, are investigations made into the origin. Now that food poisoning is notifiable, more cases and more outbreaks are thoroughly analysed.

To ptomaines have been assigned the chief cause, not only of the harmful effects resulting from the ingestion of tainted meats, but of food poisoning generally, and in consequence it has been difficult to eradicate the indiscriminately applied term 'ptomaine' poisoning. Proof has been definitely established that these putrefactive alkaloids are not present in the early stages of decomposition and are only formed when putrefaction has advanced to such a degree that the food becomes repulsive.

Substantiation of this is found in an address given before the Canned Food Section of the London Chamber of Commerce in 1922 by the late Sir William Willcox, who said : "The idea that food poisoning is due to ptomaines is quite exploded. I have made a very large number of analyses in fatal cases of poisoning and suspected poisoning ; but although I searched most minutely for all signs of alkaloidal poisons, ptomaines, and so on, unless there are some genuine chemical poison there, my efforts to find these poisons failed. I used not to succeed in finding ptomaines in the viscera which were examined, though many of them were of an extremely advanced nature as regards decomposition which had occurred. So that we can dismiss these ptomaines as the cause of food poisoning."

Rapid advances made in bacteriology and pathology furnish conclusive proof that the majority of cases of food poisoning (apart from non-bacterial food poisoning) are due to infection of the human subject by pathogenic bacilli (Salmonella) together with the toxins they manufacture. The term 'ptomaine' poisoning used in connection with food poisoning, therefore, is misleading and should be discarded in all scientific literature.

The provision of an attractive uncontaminated and unadulterated food supply is a problem of vital importance and one that has never excited so much interest in the medical profession, Government departments, public health officials, educational authorities and food manufacturers as it has during the past few years. Food is now prepared, preserved and manufactured in immense quantities by various methods and processes, often by massed production. Machinery has to a large extent replaced manual labour. Food products are frequently transported long distances in a variety of vehicles under varying conditions and are handled by a considerable number of persons before finally reaching the consumer. Thus they are exposed to contamination of all descriptions through carelessness or ignorance.

In recent years, however, there has been an important metamorphosis. The major portion of our food supply has been beyond criticism or suspicion. This is attributable not merely to legislation, which exacts in every way higher standards for products and manufacture, but to a genuine desire on the part of manufacturers, canners and traders to place on the market clean, wholesome food. Through their various trade organisations, by bacteriological and chemical research and other means, marked progress has been made in manufacture, preservation, storage,

transportation and distribution. Control of bacteria in food is now the aim of a large number of industries. This is accomplished by such means of pasteurisation, processing, the use of harmless preservatives, refrigeration, quick freezing, etc. The safeguarding and controlling of our food supplies goes to the very root of public health, and it is only by investigation and elucidation of the many difficulties associated with food poisoning, as briefly referred to above and amplified at some length in this work, that we have been able to make material and satisfactory progress towards the solution of a big problem, fraught as it is with innumerable complexities.

CHAPTER II

# HISTORICAL

From time immemorial food has been recognised as a cause of disease. Down through the ages man has gained considerable knowledge—often unpleasant or painful—as to what is fit and what is not fit to eat. Only in comparatively recent years have investigations been made and definite information obtained as to the origin and nature of the disease-producing properties associated with certain foods.

Meat, frequently the cause of outbreaks of illness, was used as an article of diet from the earliest times. Researches of geologists proved that prehistoric man lived partly on the flesh of animals. The higher hieroglyphics of the Egyptians revealed that meat and meat foods entered largely into the dietary of the ancient nations, and regulations regarding their use were introduced and officially enforced. Even in those far-off days it was recognised that animals which had died a natural death, or were killed "to save their lives," were unfit for human consumption.

Food poisoning, which was mentioned in the ancient writings of Hippocrates, Horace, Ovid and other philosophers, was of a somewhat different nature: it resulted from the accidental consumption of poisonous fungi, herbs or plants.

Records tell us that the Greek poet Euripides lost his wife, daughter and two sons, who during his absence had eaten poisonous fungi in mistake for the edible variety.

Theophrastus (300 B.C.), in his history of plants, makes several references to poisons, and records that these were sometimes added to food with criminal intent or for monetary greed. Zenophon (400 B.C.) remarks that the addition of poison to food and drink was so common amongst the Medes that it was customary for the cup-bearers to taste the wine before it was offered to the King. In the middle Ages intentional poisoning was so common that official food tasters were appointed.

During the Roman period oysters were used by Empresses, who were not the most devoted or virtuous of wives, as easy and agreeable agents in which to administer poison to their husbands or lovers. Historical records mention a number of interesting

incidents in which food was adulterated in Roman, Grecian and early English times.

Adulteration of food was practised with impunity. Sick animals were slaughtered and the diseased meat disguised or treated with preservatives and sold as sound food; the result can be well imagined. Cleanliness in slaughter-houses and premises where food was prepared was unheard of.

During the early part of the 19th century investigators of cases of food poisoning (especially meat) assigned their cause to chemical poisons in decomposed food; later, however, they were attributed to putrefactive alkaloids (ptomaines). Such outbreaks were not associated with any bacterial theories.

Albert von Haller made the first scientific observations and experiments relating to the effects of decomposed protein substances upon animals. He injected aqueous extracts of putrid meat and blood into their circulations, which caused symptoms resembling those seen in septic diseases. Experimental work on these lines was also carried out by Gaspard (1882–4) and Magendies (1823) and aroused great interest.

Panum (1856), a Kiel professor, attempted to disclose the nature of the septic poison. He demonstrated that the poisonous qualities exhibited by putrid fish were of a chemical nature and undestroyed by boiling. Bergmann and Schmiedeberg (1868) believed that the active poison was a substance they termed 'sepsin.' Later, more extensive studies were made upon the poisons in decomposed food, especially putrefying meat, and upon their effects on animals. This resulted in the publication of voluminous literature on the subject, amongst which were the monographs by Hiller (1879) and Gussenbauer (1882). Putrefactive alkaloids designated 'ptomaines' by Francesco Selmi (1872), the Italian chemist, were isolated by Nencki in 1876. In 1882–9, Brieger, Ladenburg, Vaughan and Novy investigated these substances and found they possessed highly poisonous properties, especially when injected into animals. Ladenburg (1883) prepared the first putrefactive alkaloid (Cadaverine) by synthetic methods, and in 1888 Vaughan and Novy compiled a work on ptomaines and leucomaines. Vaughan (1884) isolated 'tyrotoxicon' (a substance closely allied to ptomaines) from cheese, which had caused symptoms of poisoning.

The ptomaine theory, although it at times caused considerable controversy amongst scientists and the medical profession, was nevertheless widely accepted for many years, and the general

presumption was that the real cause of food poisoning had been discovered.

It may be mentioned in passing that it was suggested by Schwaun (1837) that putridity was really a biological process; this was confirmed by Pasteur in 1863.

The works of Vaillard (1902), Fornario (1906), Cathcart (1906) and other observers have since proved that these substances were comparatively non-toxic to experimental animals except when administered in excessively large doses, far larger than ever likely to be ingested under natural conditions. Also that ptomaìnes were not present in food until it had reached an advanced stage of decomposition when it would be repugnant in appearance and nauseating to the normal senses. Moreover, cases of food poisoning often resulted from the consumption of meat which showed no sign of decomposition and was normal in appearance.

Savage (1921) studied the relation of putrid food to illness. This was his opinion on the subject:

"The view which credits decomposed food with toxic properties largely rests upon a misconception due to the isolation of non-specific poisonous bodies called ptomaines from decomposing food, and then assuming that these bodies which are toxic by ingestion, and not at all, or to a very limited extent by feeding, are the cause of food poisoning. . . . I have fed a series of kittens with extremely putrid mixtures of canned meat and fish over long periods and without demonstrating any definite signs of toxicity. I am unaware of, and have been quite unable to find, any evidence in favour of the popular conception as to the great toxicity of incipiently putrid food or even definitely decomposed food; . . . there is no evidence of any scientific value that the general public runs any risk of illness from this source."

Tanner (1933) summarised the objections to ptomaine poisoning as a cause of illness as follows:

"1. Foods which would cause it would have to be in the later stages of decomposition, since presence of ptomaines is related to putrefaction. Most people would refuse to partake of such food.

"2. Some foods are purposely putrefied in order to improve their flavour. Such is the case with cheese, and even with meat, although in the latter case it is not carried as far as in the former. The Chinese also allow eggs to age.

"3. The toxicity of ptomaines isolated from putrefied foods has not been satisfactorily established.

"4. Symptoms of ptomaine poisoning are too inconclusive and resemble those caused by toxins formed, for instance, by members of the Salmonella group.

"5. Investigation of outbreaks of illness at first supposed to have been caused by ptomaines, has revealed more satisfactory explanations (botulism, Salmonella toxins, etc.).

"6. If ptomaines were responsible for illness, many of us would be ill much of the time. It would be difficult to avoid foods which did not contain ptomaines as they are now conceived in the minds of many."

Bollinger (1876–80) collected literature on the subject and drew attention to the relationship between meat poisoning outbreaks and the septic, pyæmic and gastro-intestinal conditions in the animals from which the meat was derived, and the heat-resisting properties of the poisons associated with such diseases which were undestroyed by cooking. He quoted eleven outbreaks of meat poisoning with about 1600 cases, the great proportion of which was of septic or pyæmic nature.

Gerlach's observations (Ostertag, 1907) upon the connection between the diseases of food animals and cases of meat poisoning are interesting. A cow sustained a severe injury to the udder from a scythe. The wound turned gangrenous and two days' later the animal was slaughtered. Although Gerlach forbade the consumption of the meat, a portion was consumed by the herder and his family. All were affected with general illness—vomiting, diarrhœa and extensive weakness.

In a further outbreak, meat from a cow which had been sick after parturition and which was emergency-slaughtered 36 hours later, was eaten by a number of persons. Forty-six became ill and 1 died. The district physician, who did not believe there was any connection between the outbreak and the consumption of the meat, ate some to prove the accuracy of his view ; he became dangerously ill.

Klein (1880) carried out some bacteriological examinations in connection with an outbreak of food poisoning (infected ham) at Welbeck, Notts. There was, however, no definite proof that the bacteria isolated caused the illness.

Probably the first bacteriological investigation into the etiology of meat poisoning was made by Johne (1884) in connection with an outbreak which occurred at Lauterbach. A number of persons were affected and 3 died. The animal (a cow) from which the meat was derived, suffered from enteritis. Johne isolated a bacillus

which was pathogenic to mice and other animals and possessed morphological characters similar to those of bacillus anthrax.

Salmon and Theobald Smith, in 1885–6, discovered the American hog organism B. choleræ-suis, afterwards named 'B. suipestifer' in 1896 by Kruse and later 'B. choleræ-suis' by Weldin (1929). The bacillus was apparently not connected at this period with any disease in man.

In May 1888 Gaertner of Jena recorded an outbreak of meat poisoning which occurred at Frankenhausen, caused by the consumption of meat from a cow emergency-slaughtered, on account of persistent diarrhœa (enteritis). The appearance of the meat was normal and the organs were not enlarged. There were 59 cases and 1 death. A man who had eaten 1½ lbs. of the meat died 36 hours later. Gaertner isolated a bacillus (which he named B. enteritidis) from the meat and blood-vessels of the cow and also from the organs of the fatal case. The organism was motile and easily stained. Dogs, cats, chickens and sparrows were immune, but mice, rabbits, guinea-pigs and goats were affected when inoculated. The bacillus during growth produced a powerful heat-resisting chemical toxin.

This discovery by Gaertner proved to be a most important landmark in the history of bacterial food poisoning, and B. enteritidis, or closely allied forms, have since been isolated during many outbreaks both in this country and abroad.

Johne (1889) demonstrated B. enteritidis in the meat from a cow which caused an outbreak of food poisoning at Cotta, Saxony, where 136 persons were affected; 4 died. The meat was eaten raw as well as cooked, thus confirming the findings of Gaertner that the toxin produced by the bacillus was not destroyed by cooking.

One of the most typical and severe outbreaks of meat poisoning caused by B. enteritidis (Gaertner) occurred at an industrial girls' school at Limerick, Ireland, in November 1909, and was investigated by McWeeney of Dublin. There were 73 cases with 9 deaths.

No information was available regarding the health of the animal from which the incriminated meat was obtained, except that it could not be fattened. It was killed in a private slaughter-house, and the meat, doubtless of poor quality, sold at a low price.

The general symptoms of the patients were acute gastro-intestinal disturbance accompanied by tenesmus and in some cases collapse.

Fig. 1.—Sir William G. Savage, M.D.

2.—Professor Theobald Smith, 1859–1934.

Fig. 3.—Professor A. Gaertner.

FIG. 4.—Professor E. J. MCWEEN 1864-1925.

FIG. 5.—EDWARD BALLARD. M.D., F.R.C.P., F.R.S., 1833–1907.

g. 6.—Prof. F. Wilbur Tanner.

Fig. 7.—P. Bruce White, B.Sc., F.R.S.

Fig. 8.—H. E. Durham, Sc.D., M.B., B.C., F.R.C.S.

Fig. 9.—Dr. Edwin Oakes Jordan, 1866–1936.

Fig. 10.
Major-General Sir Wilfred W. O. Beveridg
K.B.E., C.B., D.S.O., M.B.

The meat (stale, but apparently unaltered) was partaken of at noon and the symptoms appeared about 6 p.m. By midnight 28 of the girls were affected. The first death occurred at 7 a.m. the next morning and 8 other children succumbed within the next 2 days. Among the 73 cases every degree of severity was observed, from a condition simulating Asiatic cholera—and which at the autopsy was characterised as 'Cholera Nostras'—to slight headache and malaise with elevation of temperature lasting a few days. There were cases which showed no symptoms at all, but which presented the typical agglutination reaction in the blood and had therefore become infected. From practically all the viscera examined, as well as the discharges from the recovering cases, a typical strain of B. enteritidis was isolated. Although the bacillus was very virulent when injected into laboratory animals, guinea-pigs fed with cultures of the organism remained alive.

McWeeney failed to infect a dog by feeding it with a large quantity of meat upon which the bacillus had been grown.

McWeeney remarks: "This severe outbreak of meat poisoning was caused partly by intoxication (cf. the short incubation period), and partly by infection (cf. cultivation of the organism from the 3 fatal and 2 of the recovering cases). The causal micro-organism was the genuine B. enteritidis of Gaertner, which must have been conveyed to the sufferers in the beef, and from the history it seems probable that the calf was sickly, and already harboured the bacillus at the time of slaughter."

Ballard (1890) compiled for the Local Government Board an important summary on the then known etiological facts in relation to food poisoning.

Basenau (1893) isolated B. morbificans bovis from the muscles and organs of a cow emergency-slaughtered on account of puerperal fever. On two subsequent occasions he isolated bacilli closely allied to this organism from animals suffering from septic disease.

In America Theobald Smith (1893) investigated the fermentation properties of B. suipestifer on different forms of sugar, and his researches established the Salmonella group of organisms.

In 1896 Achard and Bensaude isolated an organism to which they gave the name 'Bacille paratyphique.' This organism, according to Boycott (1911), was Salmonelli schottmulleri (B. paratyphosus B.).

Durham (England) and de Nobele (Belgium), working independently in 1908, described a bacillus which they had isolated from patients suffering from meat poisoning. This bacillus, which was

closely related to B. enteritidis, they designated B. aertrycke after the name of the Belgian village where the outbreak occurred. The discovery proved to be of paramount importance, as B. aertrycke has proved to be the causal organism in a very large number of cases of food infection in this country.

B. aertrycke is now often designated B. typhi-murium, this being the name given to an organism isolated by Loeffler (1892) from a mouse epizootic and found to be identical with B. aertrycke. It is frequently referred to in German literature as the Breslau bacillus.

At this time it was definitely recognised that the B. enteritidis type was serologically distinct from other strains as B. suipestifer and B. paratyphosus B.

Savage (1913) remarks : " We owe an important advance in the bacteriological study of food infections to Durham, who demonstrated in 1898 that by the use of the agglutination tests the bacilli isolated from food-poisoning outbreaks hitherto all indistinguishable, could be separated into at least two distinct groups. He also drew attention to the diagnostic value of the examination of the sera of patients suffering from food poisoning."

The work of various observers, including Bainbridge and Boycott, afterwards placed the differentiation and classification on a more sound basis.

Schottmüller (1900) showed that two distinct types of paratyphoid bacilli existed ; these were afterwards named B. paratyphosus ' A ' and ' B ' respectively.

Savage (1909) concluded that B. aertrycke and B. paratyphosus ' B ' were serologically distinct, and in 1910 Bainbridge and O'Brien carried out agglutination and absorption tests and came to the conclusion that B. suipestifer and B. paratyphosus ' B ' were separate organisms and that B. aertrycke strains were identical with B. suipestifer, but in 1912 it was recognised that these two strains were not clearly differentiated.

In the Local Government Board Medical Officer's reports from 1906 to 1910 Savage reported on the following important subjects :

1. The distribution of the Gaertner group in the animal intestine.

2. The Gaertner group of bacilli in prepared meats and allied foods.

During the period 1909–23, the Salmonella group of organisms (designated ' Salmonella ' by Lignières in honour of Dr. Salmon who discovered the hog cholera bacillus), a sub-group of the typho-coli group, received a good deal of attention by various observers, both as regards their relationship to one another and their significance in certain illnesses caused by infected food. About this time considerable confusion existed in Europe and America as to which organisms comprised the Salmonella group. Much valuable research on the differentiation and classification of the various strains was carried out by Bainbridge (1909–11), Bruce White (1925–6), Boycott (1906), O'Brien (1911), and Savage (1925).

In America a vast amount of important classificatory work on serological lines was carried out by Jordan (1917), Krumweide (1918), Kohn (1918) and Valentine (1918).

Schütze (1915–20) by means of absorption tests demonstrated the existence of two serological ' aertrycke ' types. These were so-called ' Mutton ' and ' Newport ' types. In 1920 Schütze published an important and advanced work on the subject which recognised a paratyphoid B. group, constituted of nine serological types : Schottmüller (B. paratyphosus ' B ' ipse), Mutton, Newport, Stanley, Binns, Arkansas, ' G,' Reading and Hirschfeld.

Hecht-Johansen (1923) published the results of his extensive study on the classification of the typhoid-paratyphoid group of bacilli.

In 1911 McWeeney published his articles on the etiology of meat poisoning. In the next year Bainbridge, in the Milroy Lectures, gave a detailed review of the whole subject, and drew attention to the importance of the rat in relation to meat poisoning, and the possibility of the infection of food by these rodents.

In a report to the Local Government Board in 1913 Savage gave a summary of the existing knowledge of bacterial food poisoning and food infections, and in 1920 the Cambridge University Press published his valuable work on " Food Poisoning and Food Infections." It included a list of British food-poisoning outbreaks from 1878 to 1918.

During the Great War (1914–18), although enormous quantities of preserved foods were consumed by the troops at home and abroad, only one outbreak of food poisoning was recorded. This was in 1918 at a base in France and was investigated by Perry and Tidy (1919). Over 1000 men were affected. The epidemic was caused by B. aertrycke and was ascribed to a human carrier.

The Ministry of Health in 1921 issued—

1. (Memo. 39, Foods) on the procedure to be taken for the investigation of outbreaks of illness suspected to be due to food poisoning, and in 1935—

2. (Memo. 188/Med.) to Medical Officers of Health (outside London).

A copy of the latter publication is appended at the end of this work.

Among the principal works published in America on food infections and intoxications are those by Jordan (1917–31), Damon (1928) and Tanner (1933).

A large number of important articles on the subject appeared from time to time in American medical publications and scientific literature.

In England, during 1925, two special reports by Savage and Bruce White were issued by the Medical Research Council on " An Investigation of the Salmonella Group, with Special Reference to Food Poisoning," and " Food Poisoning, a Study of 100 Recent Outbreaks."

These were followed, in 1926, by " Further Studies of the Salmonella Group," by Bruce White.

The publication of these very important and comprehensive studies was another landmark in the history of food poisoning, adding as they did materially to the knowledge on the subject. The reports dealt with the identification and classification of the organisms of the Salmonella group and the physiological effects produced in animals by the results of Salmonella infections: they also described the results of detailed investigations, epidemiological and bacteriological, of 100 actual outbreaks of food poisoning in this country.

In America, during the past few years, several outbreaks of illness have been caused by the consumption of certain foods—mostly milk products, particularly cream cakes, custards, or puffs, etc., infected by a toxigenic staphylococcus.

The experimental evidence that certain staphylococci produce gastric irritation has been provided mainly by Dack, Jordan and their colleagues. Jordan in summarising the information expressed the opinion that probably many outbreaks due to staphylococci have been overlooked. These organisms are widespread in nature and consequently this opens up very considerable possibilities as a cause of obscure outbreaks.

Savage (1932) delivered the Sedgwick Memorial Lecture in America on " Some Problems of Salmonella Food Poisoning."

In February 1940 he opened an important discussion on ' Salmonella Infections ' before the Royal Society of Medicine, London, in which he said : " We have still a long way to go before we can effectively prevent the pathological manifestations of the Salmonella group in man and animals. A potent weapon is an accurate knowledge of the distribution in nature of the various types and of their specialised pathological activities."

It may be of interest in passing to mention that an entirely new English translation (edited by Dunlop Young) of an up-to-date edition of " Ostertag's Meat Inspection " was published in 1934. The history of meat poisoning in Germany is given in detail.

In November 1940 the Ministry of Health issued an important memorandum (Cir. 2198 ; 25.11.40) to Sanitary Authorities on the subject of " Precautions against the Spread of Alimentary Infections." The memorandum reminds Local Authorities of the measures which can usefully be taken to protect the public against the spread of the diseases commonly conveyed by food, i.e. diseases of the enteric group (typhoid and paratyphoid fevers), dysentery, food poisoning and intestinal parasitism.

During recent years the subject of food infection and intoxication has received much attention in the annual reports (On the State of the Public Health) of the Chief Medical Officer of the Ministry of Health. The numbers and particulars of the various outbreaks that have occurred during each year are given, together with other interesting and valuable information and advice.

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## CHAPTER III

# BACTERIAL FOOD POISONING

## (Food-borne Infections and Intoxications)

This type of food poisoning (apart from botulism) signifies illness due to the ingestion of some particular article of food which contains either pathogenic living bacilli capable of setting up acute inflammation of the alimentary tract, i.e. ‘Food Infection’ or irritative poisonous substances (toxins) only, which have been manufactured by the rapid multiplication of various types of bacilli in the food prior to ingestion, i.e. ‘Food Intoxication.’ These toxins retain their potency even after exposure to temperatures sufficiently high to destroy the bacteria producing them.

### Causation—The Salmonella Group of Bacilli

In recent years the numerous investigations into food-poisoning outbreaks, together with the intensive studies of the organisms isolated by Savage, Bruce White, Scott and other workers in this country, Jordan and his colleagues in the United States and Kauffmann in Germany, have demonstrated that certain recognised types of Salmonella are the common cause of these outbreaks. There are now over eighty distinct types of these organisms, and probably more than thirty are a cause of food poisoning. In addition, certain organisms of the dysentery group, i.e. B. sonnei and B. flexneri, also, at times, may produce the illness. The work of Bruce White, Kauffmann and others have provided a reliable basis of classification.

The table on p. 16 shows the disease-producing rôle of the most important of the Salmonella group.

The organisms of the group (p. 16), which can be distinguished by cultural or serological tests, do not form spores and have no special resistance to heat. They multiply rapidly upon meat or other suitable foodstuffs, producing poisonous substances (toxins). Some of the bacilli can be isolated from animals which are apparently free from disease, but evidence favours the view that they are only present in the carrier state, usually as the result of a previous active infection.

The table on p. 17, compiled from the annual reports of the Chief Medical Officer of the Ministry of Health, 1931–8, shows the

number of outbreaks of food poisoning due to Salmonella infections.

| Type of Organism. | Disease-producing Rôle. | |
|---|---|---|
| | Man. | Animals. |
| B. paratyphosus 'A.' | Paratyphoid fever. | Not found. |
| B. paratyphosus 'B.' | Paratyphoid fever, probably never food poisoning. | Not found. |
| B. enteritidis (Gaertner-Jena). | Gastro-enteritis of food-poisoning type. Occasionally septicæmia. | Disease in cows and calves. Epidemics in rats and sometimes disease in pigs, ducks and other animals. |
| B. enteritidis (Dublin). | Gastro-enteritis, septicæmia, continued fever. | General infection. |
| B. aertrycke. | Gastro-enteritis. Sporadic cases of illness occasionally. Quite undifferentiated. | Cause of enteritis in mice (B. typimurium), guinea-pigs and other rodents. Parrots and other birds. Occasionally in pigs and a cause of calf enteritis. |
| B. suipestifer. | Gastro-enteritis or septicæmia. | General infection. Secondary invader in pigs in hog cholera. |
| B. paratyphoid C. | Enteric type. | Not found. |
| B. Newport. | Food poisoning. Sporadic cases of illness. | Dogs suffering from enteritis, otherwise unknown in animals. |
| B. Thompson. | Food poisoning. | Doubtful isolation. |
| B. Derby. | Food poisoning. | Pigs, exact disease-producing *rôle* unknown. |
| B. morbificans bovis. | Food poisoning. | Original strain from cows suffering from puerperal metritis. |
| B. abortus equi. | Not found. | Abortion in mares. |
| B. Stanley. | Food poisoning. | Not yet isolated. |

Outbreaks due to members of the Salmonella group are met with in many parts of the world. They are common in Europe and America, and cases have been reported from the widely

| Year. | Number of Outbreaks of Suspected Food Poisoning. | Outbreaks Due to Salmonella Poisoning. | Cases. | Deaths. | Bacterial Types Isolated and Identified. |
|---|---|---|---|---|---|
| 1931 | 43 | 18 | 425 | 10 | Aertrycke 13.<br>Enteritidis 2.<br>Thompson 1.<br>Dublin 1.<br>Morbificans bovis 1. |
| 1932 | 55 | 24 | 186 | 8 | Aertrycke 6.<br>Newport 2.<br>Thompson 1.<br>Enteritidis 2.<br>Unidentified 3. |
| 1933 | 75 | 32 | 512 | 15 | Aertrycke 15.<br>Enteritidis 6.<br>Newport 3.<br>Thompson 4.<br>Suipestifer 2.<br>Unidentified 2. |
| 1934 | 58 | 43 | 125 | 6 | Aertrycke 22.<br>Newport 8.<br>Enteritidis 4.<br>Potsdam 3.<br>Eastbourne 2.<br>Thompson 6.<br>Suipestifer 2.<br>Dublin 1. |
| 1935 | 137 | 46 | 695 | 23 | Derby 1.<br>Newcastle 1.<br>Suipestifer (European 1).<br>Suipestifer (American 1).<br>Aertrycke 29.<br>Enteritidis 8.<br>Thompson 3.<br>Morbificans bovis 2. |
| 1936 | 82 | 19 | 224 | 3 | Aertrycke 11.<br>Enteritidis 3.<br>Thompson 2.<br>Newport 1.<br>Dublin 1.<br>London 1. |
| 1937 | 94 | 45 | 601 | 24 | Aertrycke 19.<br>Enteritidis 9.<br>Newport 8.<br>Thompson 5.<br>Morbificans bovis 1. |
| 1938 | 156 | 72 | 330 | 8 | Aertrycke 37.<br>Thompson 11.<br>Enteritidis 7.<br>Newport 5.<br>Suipestifer 2.<br>Dublin 2.<br>Stanley 1. |

2 The figures for 1939 and 1940 are not available.

scattered areas of Asia and Africa. In Great Britain food poisoning of the gastro-intestinal type is usually due either to Salmonella infection or some form of toxin outbreak. Occasionally, however, other organisms are associated.

The organism most commonly isolated in British outbreaks is B. aertrycke.[1]

During the years 1923–33, the Ministry of Health found B. aertrycke in 110 out of 186 outbreaks; and from 1934–8, 118 out of 225 outbreaks.

The next organism most frequently isolated is B. enteritidis.[1] Outbreaks due to this bacillus appear to be more common in countries other than England and tend to be more severe. Bruce White (1929) differentiated 'Dublin' from the ordinary B. enteritidis, his strain coming from a fatal case of continued fever in Dublin. After its differentiation it was possible to show that it was this type which was especially associated with calves (calf dysentery) and cattle. Knoth (1936) examining meat from slaughtered animals found that of 538 strains from calves, 506 were Dublin types and 17 out of 18 from adult cattle were Dublin. This has been confirmed by several other observers.

Savage (1940), in a discussion on Salmonella infection before the Royal Society of Medicine, introduced the undermentioned table (p. 19), showing infections with the Dublin type.

He said: "Since the differentiation of Dublin it has been found to be the cause of human infections in a number of cases. No doubt a number of outbreaks due to B. enteritidis, especially from milk, were due to Dublin, but as the strains have not survived to be differentiated we have no accurate knowledge."

## Toxin Manufacturing Properties

During recent years evidence has accumulated, showing that many outbreaks have been due to undestroyed poisonous substances elaborated by certain organisms including the Salmonella group. This was due to the fact that intensive bacteriological research failed to reveal the presence of the causative organisms. The extremely short incubation period (2 to 4 hours or even less), together with the very severe symptoms, suggested the action of a preformed toxic substance in the food ingested, more especially in the case of canned foods.

Topley and Wilson (1936) remark: "It was supposed that the organisms had multiplied in the food prior to its consumption

[1] See illustrations, facing page 52.

and had formed thermostable toxic substances. The subsequent cooking to which the food was exposed destroyed the organisms themselves, but did not seriously affect their toxic products, which

Human Infections with the Dublin Type

| Place. | Reference. | Particulars. |
|---|---|---|
| Dublin. | Bruce White, 1929. | Pyelitis kidney and continued fever; single case. |
| Aberdeen. | Smith and Scott, 1930. | Three unconnected cases of continued fever. All positive blood cultures. All recovered. |
| Aberdeen. | J. Smith, 1933. | Three unconnected cases (two infants, one 5 years); one septicæmia and mastoiditis, blood positive, fatal. One gastro-intestinal disturbance, blood negative, recovery. One meningitis, fatal; bacilli in cerebrospinal fluid. |
| Aalborg (Denmark). | Grimsted, 1923. | About 95 cases of acute gastro-enteritis at Aalborg Hospital. No deaths. Vehicle milk. Diseased cow which died and B. paracoli isolated from spleen and udder. Same organism in fæces of cases. |
| St. Pancras, London. | Ministry of Health Report, 1928. | Cases 22, no deaths. Vehicle junket. Suggested that was locally infected but information indefinite. Dublin isolated from fæces of cases. |
| Dundee, 1927. | Tullock, 1939. | About 280 cases of acute gastro-enteritis, no deaths. Vehicle milk. Dublin type from fæces and from internal organs of a diseased cow. |
| Wilton, 1936. | Conybeare and Thornton, 1938. | Over 100 cases of gastro-enteritis in children, no deaths. Vehicle milk. Fæces examined late and negative. Milk contained Dublin, and this isolated from dung of cow with high titre. |
| S. Africa, 1938. | Henning, 1938. | Ten natives ate sick calf under-cooked. All suffered from food poisoning and one died. Dublin isolated from fatal case. |

were therefore able to give rise to food poisoning on ingestion. No adequate confirmatory evidence of the formation of specific exotoxins by members of the Salmonella group was forthcoming and

the balance of evidence appeared to be against this view. . . . Summarising, we may say that evidence has been accumulating in the past few years to show that many of the 'toxin' outbreaks of food poisoning are due to the production of toxic substances in the food prior to its consumption. These substances, the exact nature of which is still unknown, are formed under suitable conditions by a number of different bacteria, of which staphylococci, streptococci, coliform, Proteus, and possibly Salmonella, organisms appear to be the most important."

Savage (1920, 1923 and 1932) advanced the toxin theory, and Savage and Bruce White in 1925, referring to outbreaks due to the presence of undestroyed Salmonella group toxins, stated: "These form a very important group, particularly in relation to canned foods. It will readily be appreciated that the furnishing of complete, or even presumptive, proof that these toxins are the cause of any outbreak is a matter of great difficulty. There are no living bacilli to isolate. Our studies on this point have been to a certain extent progressive, and for the later outbreaks improved methods have enabled us to furnish proofs of a more conclusive nature than we were able to do for many of the earlier outbreaks."

This toxin hypothesis at times created a considerable amount of controversy both in this country and abroad.

Savage and Bruce White (1925) studied the methods of action of strains of the Salmonella group upon the alimentary tract and demonstrated the presence of a powerful irritant, both in boiled and unboiled cultures, which acts rapidly and intensibly upon the mucous membrane of the stomach of young rabbits and was most readily demonstrated in those types within the group which were responsible for food poisoning. Later experiments showed that it was possible to produce toxic effects upon mice when fed with Salmonella strains grown in certain media.

It may be of interest to mention the following outbreak, which occurred at Edinburgh in 1926, as illustrating this type of toxin poisoning. Three persons consumed a mutton stew and were attacked, after a very short incubation period, with acute food-poisoning symptoms. From the stew no "living Salmonella bacilli" could be isolated, but from part of the mutton not used to make the stew, a Salmonella strain was isolated which was pathogenic to guinea-pigs.

Apparently little is known, however, of the exact nature and mode of origin of these poisonous substances, whether they are specific bodies elaborated by bacteria or whether they represent



5

the breakdown products of dead organisms. They are not always poisonous when fed to experimental animals.

## Resistance to Heat

The remarkable heat-stable properties of these poisonous substances (Cathcart found that B. enteritidis toxin withstood heating to 100° C. for 30 minutes) have considerable bearing on the processing of canned foods, especially in the United States where the subject has been under active investigation, on account of its importance to the food preservation industry.

Savage (1932) called attention to "the close association of this type of food poisoning with canned foods—that is, foods strongly heated after they are put into the tin. The temperatures used (100° C. or above) are adequate to kill non-sporing bacilli, but Salmonella toxins can survive these temperatures. Assuming specific infection, before canning, of a portion of the food, the conditions actually found—that is, a food perfectly sound physically, freedom from living pathogenic bacilli, the presence of resistant toxins,—are just those one would expect."

A considerable amount of research and experimental work has been carried out in this country, in America and in Germany to ascertain the thermal destruction point of toxins. This varies through a wide range of temperatures and is dependent, moreover, on several factors, including the character (size of food particles) and composition of the contents of the can, the hydrogen-ion concentration and the nature of the toxin itself. In canned foods the heat penetrates to the centre of the contents by convection and conduction, but the character of the food greatly affects the convection currents. In thick and solid substances such as meat, the heat, being by conduction, penetrates very slowly. Therefore it is necessary that canned foods should be submitted to a sufficiently high temperature for the required length of time to be certain of the destruction of any bacterial toxins that might be present. It is also essential in processing that no over-heating takes place, otherwise the food may be spoiled.

In this connection some interesting investigations have demonstrated that meat, in particular, is a poor conductor of heat.

Perroncito studied the heat penetration of a boiled ham A ham of about 6 kilos weight (13 lbs.) was placed in cold water which was raised to boiling-point. The water boiled when the interior of the ham was only 25° C. (77° F.). After 35 minutes the temperature was 35°–40° C. (95°–104° F.). After 2 hours the temperature

in different parts of the interior was 46°, 55°, 58°, 62°, 64° and 67° C. (152·6° F.). A larger ham, weighing about 8 kilos (16 lbs.), treated in the same way, only showed an interior temperature of 44·5° C. after $2\frac{1}{2}$ hours, while after $3\frac{1}{2}$ hours the temperature varied from 62°–84° C. (183·2° F.) in different parts.

Beveridge and Fawcus (1908) carried out some experiments on the penetration of heat into the substance of meat in cans. They found that when a tin was simply boiled in water, the centre of the meat did not reach 100° C. even after 5 hours, and that with higher temperatures, the undermentioned results were recorded :

| Outside Temperature. | Size of Tin. | Time Taken by Central Thermometer to Reach | | Number of Experiments. |
|---|---|---|---|---|
| | | 100° C. | 105° C. | |
| 107° C. | 1 lb. | 58 minutes | 80 minutes | Average of 5 |
| 107° C. | 2 lbs. | 95 ,, | 123 ,, | ,, 5 |
| 120° C. | 1 lb. | 22 ,, | 24·4 ,, | ,, 5 |
| 120° C. | 2 lbs. | 28 ,, | 36·2 ,, | ,, 5 |
| 130° C. | 2 lbs. | 17 ,, | 22 ,, | ,, 2 |

With larger tins the rate and time of heat penetration would be considerably longer.

In liquid foods, such as soups and beverages, the heat being by convection, penetrates rapidly and, providing they are boiled for a sufficient length of time, destroys infectivity. This is well illustrated in an outbreak at Newcastle (1913) where 523 people consumed milk infected by B. enteritidis, and not a single person who drank the milk after it had been boiled was infected.

## Staphylococci, Streptococci, and other Organisms in relation to Food Poisoning

Regarding poisonous substances produced in food by other than Salmonella organisms, the research and experimental work in the United States by Dack and his colleagues (1930), Jordan (1930–1), Jordan and Hall (1931), Jordan and Burrows (1933–4–5), Dolman (1934) and other workers, showed that certain strains of staphylococcus produced noxious substances which were responsible for a considerable number of food-poisoning outbreaks. Proof was obtained by feeding sterile filtrates to human volunteers ; they caused gastro-enteritis with severe collapse.

There appears to be no reliable information as to why only some strains of staphylococcus produce these endotoxins, or the conditions which induce certain strains to develop this property. Jordan points out that it is as yet uncertain how frequent the staphylococcus type of food poisoning really is. Probably it has occurred much more often than generally recognised. The incubation period is short and the symptoms similar to Salmonella poisoning. The toxic substance is not completely destroyed by exposure for 30 minutes to 100° C.

" During recent years a moderate number of outbreaks of food poisoning have been shown to be caused by certain staphylococci which produce an endotoxin pathogenic to man ; as the favourite vehicle is cream cakes and sometimes milk, it is possible that the milk is not only the vehicle, but that the organisms may be derived from an udder infection. This possibility cannot therefore be ignored. . . . Staphylococci are so widespread in distribution that it is obvious that only a few specialised strain are of this type " (Savage, 1941).

The Chief Medical Officer of the Ministry of Health in his Annual Report for 1936, in recording toxin outbreaks, says : " Of the remainder, 26 were regarded, from the clinical course (very acute gastritis and enteritis within 3 hours of a suspected meal, followed by rapid recovery), as due to a ' toxin ' already elaborated in the food as a result of bacterial growth and not due to Salmonella infection. From the suspected food, cultures of staphylococci, usually aureus, were isolated in 10 instances in such numbers as to suggest that they had produced the gastro-intestinal irritant responsible for the symptoms. In one instance (canned tomatoes) heat-resistant streptococci of the fæcalis type (enterococci) were present in great abundance and almost pure culture."

An interesting and typical outbreak of staphylococcal food poisoning occurred at Leicester in 1942 (Lawrence).

" In the afternoon and evening of October 31, 1942, nine people in four separate families were taken violently ill with nausea and vomiting. In some cases this was followed by diarrhœa and prostration. One person had a slight hæmatemesis and one a rigor, but recovery was rapid and they were all more or less well in 24 hours. The outbreak was at once traced to some American chopped ham bought the same day and eaten for lunch and tea, symptoms coming on from 1½ to 2½ hours later. No one in the four families concerned who ate the ham escaped the illness, and symptoms were confined to these alone.

"A sample of the ham from one of the families was brought up for bacteriological examination the following morning—about 18 hours after the onset of the outbreak and 24 hours after the tin from which the ham came was opened. It appeared clean and smelt fresh, but was found to contain over 100,000,000 coagulase-positive *Staphylococcus aureus* per gram. Apart from a few coagulase-negative *Staphylococcus albus*, these were the only organisms isolated.

"The history of the particular tin concerned is :

Oct. 28. Tin arrived at the shop, together with eleven similar tins from the same wholesale distributor.

Oct. 31.

11.30 a.m. Tin opened and the contents removed, wrapped up in paper and placed on a shelf in the shop. The manager noticed that the small amount of jelly surrounding the ham in the tin was a little watery, but this was also noticed in tins unassociated with food poisoning, which were subsequently shown to be uncontaminated.

1.0—3.0 p.m. Portions of the ham sliced off the main block by manager and sold to the four families concerned in the outbreak, but to no one else.

1.30 p.m. Portion bought by first family was eaten by 4 members of the household for lunch. Symptoms came on about 2½ hours later.

4.0 p.m. Very small portions ('about the size of a half-crown') cut off the main block and eaten at once by the manager, his wife and one assistant. These persons had no symptoms whatever.

4.30—5.30 p.m. Portions sold to the other three families were eaten by certain members of these families.

"Specimens of fæces from all those who were ill were examined as soon as possible, but were in no case found to contain any staphylococci or other pathogenic organisms. Samples of the ham sold to two of the other affected families were examined and found to contain about the same number of coagulase-positive *Staphylococcus aureus* as the first sample. The unsold block of ham was also examined. The outside portion was found to contain about 400,000,000 coagulase-positive *Staphylococcus aureus* per gram, and numerous gram-positive cocci could be seen in a direct smear ; the inside, however, was relatively uncontaminated, containing only about 100 organisms per gram and no *Staphylococcus aureus*.

The trace of jelly remaining in the tin, although giving a high count of about 1,000,000 organisms per gram, contained no *Staphylococcus aureus.* A second tin of the same batch was opened with sterile precautions and even the outside of the meat contained only about 1,000 organisms per gram and no *Staphylococcus aureus.*

" Conclusions :

" This outbreak has the features—short incubation time and very rapid recovery—usually associated with food poisoning of the toxin type, and there can be little doubt that *Staphylococcus aureus*, present in such large numbers, was the cause. The single most important fact appears to be the short time (in one case only 2 hours) elapsing between the tin being opened and the ham being eaten. This, by itself, would seem to rule out the possibility of toxin being formed after the opening of tin. Moreover, there seems to be no obvious way in which the ham could have been contaminated after opening ; careful enquiry showed that neither the manager nor any worker in the shop was suffering from any boil or form of skin infection which might have acted as a source of pathogenic staphylococci.

" The failure to isolate *Staphylococcus aureus* from the inside of the tin is admittedly difficult to account for.[1] The absence of symptoms in the three shop workers who ate the ham is not, however, so surprising, since they are known to have eaten very small quantities. An alternative explanation for both these facts is that the contamination was not uniform, so that parts of the meat remained free both from pathogenic staphylococci and toxin.

" The bulk of the evidence strongly suggests that the ham was contaminated during the canning process.

" It is unfortunate that there appear to be no means of recognising on the spot food that has been heavily contaminated with staphylococci and is unfit for consumption. In this particular instance the ham was perfectly normal in appearance, and had a delicious and appetising smell."

In America Jordan and Burrows (1935) also have shown that certain freshly isolated strains of streptococci, B. coli and B. proteus occasionally produce toxins (termed enterotoxins). They found that at least four strains of B. proteus possessed this property. These investigations considerably widen the bacterial possibilities responsible for outbreaks of food poisoning.

[1] This might be accounted for by the fact that the ham is wrapped in paper in the tin (Lawrence).

## The Proteus Family

These are widely distributed in nature and can be isolated from raw meat which has been left a few hours in a warm place. As a result of their growth decomposition sets in and later the meat becomes soft and slimy.

Outbreaks of food poisoning ascribed to B. proteus have been described by Levy (1894), Wesenberg (1898), Glucksmann (1899), Schumburg (1902), Ohlmacher (1902), Pergola (1910), Mandel (1912) and many others.

Later, Bengston (1919), Savage (1920) and Tanner (1933) carefully studied many of the recorded outbreaks regarded as due to B. proteus and reported that in none of these was it established that this bacillus was ætiologically concerned.[1]

Jordan and Burrows (1935) over a period of ten years prepared broth filtrates of proteus strains, many of which were regarded as the cause of outbreaks, and fed them to human volunteers without any observable effects. From the evidence available, it would appear that these special strains of B. proteus are very rare and consequently cases of food poisoning due to them are not frequent.

It may be of interest to quote from an editorial article on this subject, which appeared in *Public Health* in April, 1941 :

" The key to the discrepancy is given by the laboratory studies (referred to) which show that only rare and special strains of B. proteus (or B. coli) have acquired the property of producing an enterotoxin. Given such a strain it may be accepted that B. proteus, like the special staphylococci, may be responsible for an attack of food poisoning. Our present knowledge, however, is to the effect that this property is much rarer with proteus strains than with staphylococci. This infrequence makes any outbreak proved to be due to B. proteus of special interest. Such a one has recently been reported by Cooper, Davies and Wiseman at Bristol. The outbreak was from brawn, the pigs' heads from which it was made being stored for three days in a brine bath, then sold to a canteen and only two days later made into brawn. The material was said to have been boiled for 3 hours. The actual number of cases is not stated, but nine persons were definitely affected, while there was an additional number of mild cases. The incubation period was 3 to 5 hours and the usual food-poisoning symptoms of vomiting, diarrhœa and abdominal pain were present, but in 5 cases there were marked collapse and cyanosis ; all patients recovered.

[1] See illustration, facing page 52.

Proteus strains were isolated from the brawn and from a number of samples of fæces. Several strains from both brawn and fæces were positive when tested for enterotoxin by the intraperitoneal kitten test. The brine pickle also yielded similar strains of Proteus vulgaris. This outbreak can be accepted as due to a Proteus vulgaris which produced enterotoxin and which was allowed to grow for a number of days in the pigs' heads before being made into brawn. Either the boiling was not boiling or there was re-infection of the made brawn, for this organism has no high resistance to heat."

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CHAPTER IV

# SEASONAL PREVALENCE OF BACTERIAL FOOD POISONING

THE seasonal variation of food poisoning due to Salmonella is probably a result of the rise in temperatures during the summer months which may favour the rapid multiplication of the bacilli in infected foods, thus producing the moderately large dose necessary to cause acute gastro-intestinal symptoms. It is possible that Salmonella organisms have greater virulence in warm weather and that susceptibility to infection by human beings may be by increased sensitiveness of the alimentary tract during the warmer months. It has also been suggested that the seasonal incidence depends to some extent on the proportions of canned and preserved food to fresh food that is eaten. The highest incidence occurs between the months of May and October with ' peak ' in July.

## Clinical Features—Incubation Period—Symptoms

The symptoms of Salmonella bacterial food poisoning vary in duration and intensity in different outbreaks. There is, however, distinct uniformity in the clinical features of all the cases.

No manifestation occur during the incubation period (i.e. the interval between the consumption of the incriminated food and onset of symptoms) which varies considerably for the reasons explained below and ranges from half an hour to 48 hours, but averages from 6 to 12 hours. These time variations are influenced by the following :

A. The ingested food may contain living bacilli ' only ' and in consequence a definite incubation period must elapse during which the organisms manufacture sufficient poisonous substances (toxins) to produce the symptoms.

B. The incriminated food may contain living bacilli together with a small amount of preformed toxin. The incubation period will then vary, being influenced by the amount of food (and toxin) consumed and the degree of bacterial contamination. The majority of food-poisoning outbreaks are of this type.

C. The ingested food may contain preformed heat-resisting toxins ' only.' In this case the incubation period will be short as

## Seasonal Prevalence of Bacterial Food Poisoning

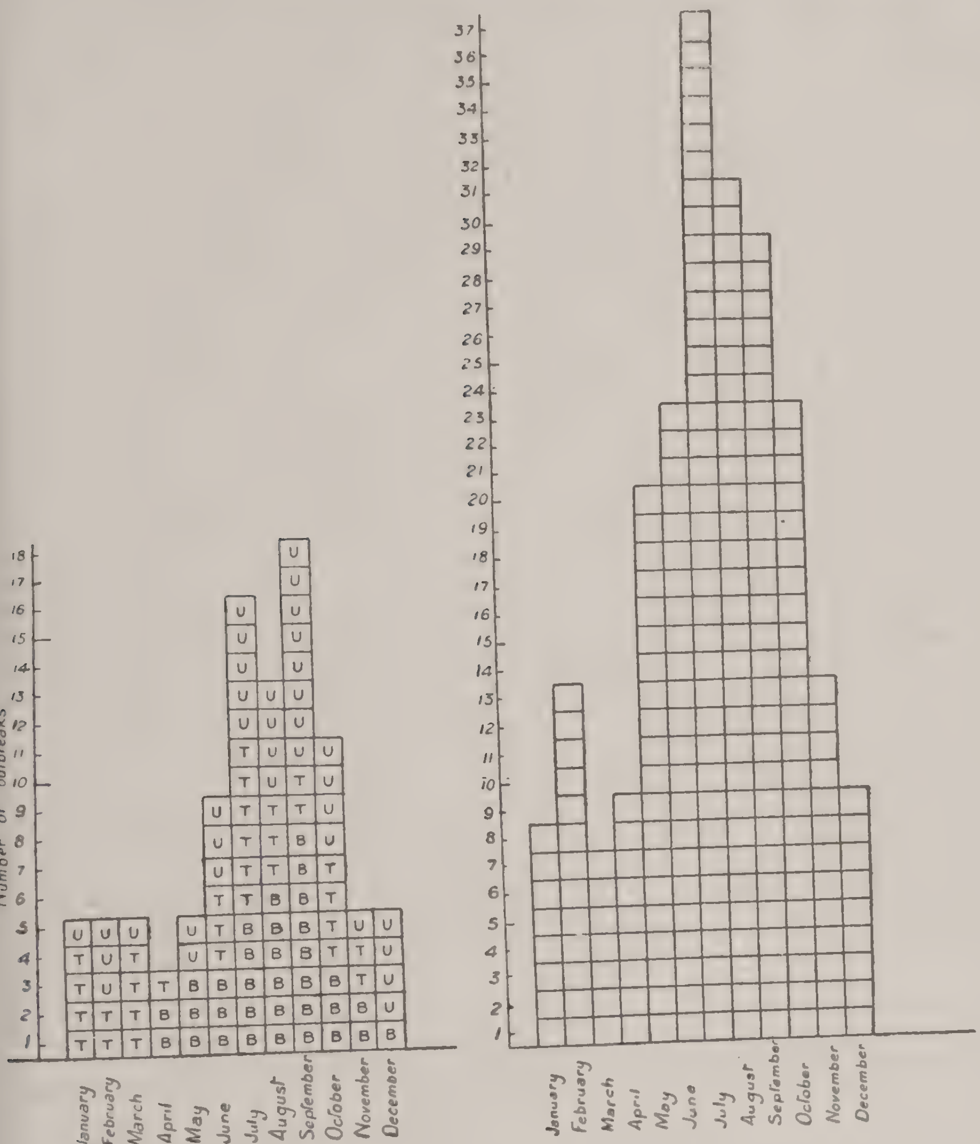


FIG. 14.

Seasonal Prevalence.
100 Outbreaks of Food Poisoning.

B. Outbreaks due to living bacilli.

T. Outbreaks due to toxins of the Salmonella group.

U. Other outbreaks probably due to bacteria but causation uncertain. (After Savage and Bruce White.)

Seasonal Prevalence.

222 Outbreaks of Food Poisoning in Great Britain and Ireland. (After Savage and Bruce White.)

the poisoning substances being all present at once produce the symptoms quickly and more markedly, but recovery is, as a rule rapid.

The onset of bacterial food poisoning is fairly sudden, usually commencing with abdominal pain. The characteristic symptoms in typical cases are invariably gastro-intestinal irritation, which may be only simple diarrhœa (with or without sickness) to severe inflammation of the alimentary tract, accompanied by vomiting (may be absent in some cases), intense abdominal pain, cramp and marked prostration : the latter is a characteristic feature and may persist long into convalescence. The diarrhœa is usually severe with repeated offensive motions. Later the stools become watery, of a greenish colour and occasionally tinged with blood. Headache is frequently present and the tongue becomes coated and breath offensive. In some instances there may be cold sweats, rigors, giddiness and pains in the back and limbs. Herpes or urticarial rashes are not uncommon, and occasionally eye symptoms (pupil irregularity) have been recorded. In severe illness collapse occurs, sometimes with fatal result, but as a rule the symptoms gradually diminish after 48 hours.

## Mortality

The mortality rate of bacterial food poisoning is distinctly low but varies in different outbreaks. In the 112 outbreaks with 9190 cases which occurred in England and recorded by Savage in 1920, it was 1·5 per cent. Some observers give an average of 1 per cent. to 4 per cent. mortality. In America Geiger (1923) estimated it at less than 0·5 per cent. Meyer (1913) in Germany recorded a death-rate of only 1 per cent. in outbreaks due to Salmonella organisms. In milk outbreaks alone Savage and Bruce White found the mortality rate to be less than 0·2 per cent. It would appear that in outbreaks due to living Salmonella organisms the mortality is established at 1·2 to 1·3 per cent., but when due to toxins is much lower, and in 31 outbreaks it was only 0·54 per cent.

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CHAPTER V

# KINDS OF FOOD THAT ACT AS VEHICLES OF INFECTION

THE suitability of a food as a medium for the multiplication of the organisms of the Salmonella group is of considerable importance. It has been observed that meat and meat products, especially made-up or manipulated foods, have proved to be the commonest classes of foodstuffs that act as vehicles of infection.

The undermentioned table, which includes 203 British outbreaks investigated by Savage and Bruce White, shows that 72 per cent. of the incriminated articles were 'made-up' or 'manipulated' foods :

| Nature of Food. | Outbreaks. | Percentage. |
|---|---|---|
| Canned meat | 31 | — |
| Canned marine products | 27 | 30·5 |
| Canned fruit | 4 | — |
| Milk | 14 | 6·9 |
| Milk products | 16 | 7·9 |
| Made-up meat | 54 | 26·6 |
| Manipulated meat | 10 | 4·9 |
| Fresh meat | 33 | 16·3 |
| Fruit and vegetables (not canned) | 8 | 3·9 |
| Other foods | 6 | 2·9 |

The following (Savage 1932) give the vehicle in 121 food-poisoning outbreaks (Great Britain, 1919–1931) associated with presence of living Salmonella strains :

| | |
|---|---|
| Milk and milk products (milk 5, cream 1, ice cream 3, junket 1, trifle 2) | 12 |
| Meat pies (pork 10, veal and ham 3, various 3) | 16 |
| Minced meat foods (minced or potted 8, brawn 6, sausages 3, stuffing 1, other 3) | 21 |
| Canned foods (beef 2, salmon 4, fruit 2) | 8 |
| Somewhat manipulated foods (salted beef 2, pressed beef 2, meat stews 2, ham 2, bacon 2) | 10 |
| Eggs (duck 6, egg sandwiches 1) | 7 |
| Fresh meat (pork 2, beef 1, mutton 2, veal 1, unspecified 2) | 8 |
| Various (fried fish 2, shell-fish 2, other 1) | 5 |
| Not ascertained with reasonable certainty | 34 |

Vehicle in 70 ' toxic ' type of food-poisoning outbreaks in Grea
Britain, 1919–1931 :

| | | *Outbreaks* |
|---|---|---|
| Tinned foods (82·9 per cent.) | | 5 |
| Beef | 26 | |
| Mutton | 1 | |
| Tongue | 9 | |
| Salmon | 13 | |
| Sardines | 2 | |
| Herrings | 2 | |
| Shell-fish | 3 | |
| Peas | 2 | |
| Made-up meat foods | | 5 |
| Fish paste | 1 | |
| Brawn | 2 | |
| Veal and ham pie | 1 | |
| Sausage | 1 | |
| Less manipulated meat foods | | 5 |
| Salted beef and pork | 1 | |
| Pickled tongue | 2 | |
| Pressed beef | 1 | |
| Frozen meat | 1 | |
| Other foods | | 2 |
| Cheese | 1 | |
| Cooked beef | 1 | |

It must be borne in mind that foodstuffs such as meat pies, potted meats, brawn, faggots, pork pies and other forms of made-up foods, which contain a considerable amount of jelly or solution of gelatin or agar-agar, after being prepared are often allowed to stand for some time to cool slowly. Such foods constitute an excellent medium for the growth of organisms which have many hours at a suitable temperature in which to multiply rapidly, and the infection is probably favoured by air currents set up in the cooling mass ; moreover, preparation and cooling may take place under conditions where infection during or after preparation is likely to occur. Lastly, though probably not of least importance, the meat used may be diseased or unsound.

It is important to remember that investigations into outbreaks of food poisoning often show that the food purchased has not been consumed in a fresh condition, or that it has been heated up on the second or third day, or that it has been placed in an oven for a short period " to prevent it going bad " and not cooked and eaten until the following day.

In one outbreak which occurred in Staffordshire in 1930, there were 8 cases and 3 deaths. The poisonous pork pie was baked and sold on 9th April but was not consumed until 13th April.

Savage (1932) says : " Again and again the facts show that the original food is harmless and the infection with the bacillus takes place on the premises. It is very significant that in many outbreaks the food sold first causes no illness, although the facts suggest that it is already infected, while the food which is sold last —that is, allowing a longer period for the multiplication of the Salmonella bacilli—is the most poisonous, causes the most severe attacks and includes the fatal cases."

A significant feature of food-poisoning attacks on the Continent is the frequency with which they have been traced to the use of raw or imperfectly cooked food.

Of 44 Continental outbreaks occurring from 1888 to 1910 41 were due to the ingestion of meat foods, these included 5 from horse flesh.

In Germany between the years 1923 and 1928, 76 outbreaks of poisoning occurred ; 4419 persons were affected and 27 died. 6·3 per cent. of the outbreaks, 79 per cent. of the cases and 57·4 per cent. of the deaths were due to the consumption of minced meat prepared from emergency-slaughtered animals.

Owing to the number of outbreaks which have been associated with cooked foods, such as meat pies, hams, *pâtés*, etc., investigations have been made by many observers from time to time both in this country, in America and in Germany to ascertain whether the temperature reached in cooking such food is sufficient to destroy any pathogenic organisms that may be present. In some experiments as to the temperature reached in baking meat pies, carried out by Delépine and Howarth (1902), in connection with an outbreak of food poisoning (meat pies) at Derby, they noted that the centre of a pie which appeared externally to be well-baked (but was really under-baked) did not exceed 47·2° C., and that the centre of a pie which was over-baked had not reached beyond 86·6° C. Furthermore, there was a difference of several degrees between the temperature of various pies. Delépine pointed out that a batch of pies prepared in a hurry might be so cooked that bacteria might continue to grow in the centre while in the oven and certainly would not be killed. He also came to the conclusion that the outbreak of illness was due to the presence in the pies of B. enteritidis (Derby), and that the meat was contaminated before it was baked.

The experiments again proved that meat is a poor conductor of heat, and demonstrated that the temperatures reached by the interior of the meat in cooking may be below that necessary to kill pathogenic organisms.

In recent years a number of outbreaks has been recorded in which Salmonella infection has been transmitted by duck eggs. During 1936 in 5 outbreaks the evidence strongly suggested a duck's egg as the source of infection ; the same cause was suspected in a small outbreak affecting 3 persons who consumed meat rissoles bound with duck's egg. All were instances of aertrycke infection and two were fatal. In all but one instance the suspected egg was imported.

## Appearance, Taste and Odour of the Incriminated Food

A popular and mistaken idea exists that food to be toxic and dangerous must be tainted or have a suspicious or disagreeable smell. Usually there is nothing in the appearance, taste or odour of the infected article to suggest that it is unfit for consumption ; in fact it generally appears quite normal. It is not surprising, therefore, to find that experienced persons are sometimes led astray. A striking and melancholy illustration of this took place in Brussels in 1896. A meat inspector examined some saveloys, which were suspected of causing illness and in the absence of any normal signs, passed them fit for consumption. By way of showing confidence in his opinion and to demonstrate their harmlessness he partook of some of them himself. He was attacked by choleraic symptoms of the severest type and died in 5 days. Van Ermengem (1906), who investigated the outbreak, isolated B. enteritidis from his viscera and from the saveloys. Altogether a large number were made from the same meat on the same day, only four of them were infected, thus illustrating that the distribution of pathogenic organisms in an article of food may be quite uneven.

Very occasionally alteration in the physical appearance of infected meat foods does occur, but this usually is only detected by a trained eye. Delépine, when investigating an outbreak at Accrington, noticed that one of the surfaces of the samples of food was smooth and was covered with a thin layer of jelly which was slightly turbid, due to numerous colonies of bacteria. The organisms which usually produce physical changes in food are those causing decomposition ; the results are so obvious as to prevent the article being consumed.

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CHAPTER VI

# POSSIBLE SOURCES AND MODES OF INFECTION

TRACING the primary source of Salmonella bacilli in food-poisoning outbreaks has always been found an exceedingly difficult problem. Although a considerable amount of information on the subject has been collected, recent knowledge attained by observers, after studying a large number of individual cases and outbreaks, has revealed that in many instances although the definite cause of the illness has been discovered, i.e. infection of the food consumed, by Salmonella organisms, the primary source of the bacilli (the actual reservoir) and the mode of transmission, thence to the food ingested (path of infection), could not be ascertained, or only incomplete information was obtainable which eventually proved of little value to the investigators in discovering these important issues. This, perhaps, was due in a measure to delay in commencing the investigations or to difficulty in obtaining the necessary material for bacteriological confirmation. Now that food poisoning has been made notifiable, doubtless unnecessary delay will be avoided.

The known ways in which food may become infected by Salmonella organisms are as follows :

A. Meat from a diseased or infected animal or passive carrier, i.e. beef, veal, mutton and pork.

B. Milk from an infected animal.

C. Infection transmitted by duck eggs.

D. Infection transmitted by rats and mice.

E. Human carriers (infected sufferer or passive carrier).

F. Possibility of flies acting as carriers of infection.

## A. Meat from a Diseased or Infected Animal

In this country the majority of the reports on food poisoning outbreaks contain little information regarding the health of the animal from which the food is derived. This shows the importance of ante-mortem and post-mortem examinations of food animals.

Savage (1932) remarks : "The only sources of infection of which we have accurate information are animals or birds either suffering from Salmonella infections or with the bacilli present in a carrier condition. In one group we can include animals used by

man for food. Calves and cattle suffer from Salmonella infections, the strain being usually B. enteritidis, less commonly B. aertrycke, while B. morbificans bovis was originally isolated in 1893 from a diseased cow. Swine infections with Salmonella bacilli are well known, either as a cause of definite enteritis or as secondary invaders in swine fever. . . . Records of Salmonella disease in sheep are scanty but several have been described, mostly due to B. aertrycke."

In the early days, Bollinger maintained that the flesh of animals suffering from septic and pyæmic diseases were unfit for human consumption. There seems little doubt that animals, especially cattle and pigs, may suffer from umbilical infections, intestinal and other diseases caused by Salmonella organisms. The disease is usually acute and often fatal, but the carcase need not necessarily be noticeably unhealthy.

A severe outbreak occurred in Uberuher, South Africa, in 1919. Of 4000 inhabitants more than half were affected. The mutton which was responsible was obtained from sheep which had gastro-enteritis. The carcasses were released for sale as, on inspection, nothing was found beyond slight reddening of the mucous membrane of the stomach and intestines. From the carcass and from the stools of infected humans the Salmonella aertrycke were isolated. Most cases occurred when food, which was not properly cooked, was consumed, and in some cases infection took place merely by handling infected meat. So that, in this case, the condition in the human beings was more of an infection than an intoxication (Fourie 1936).

In the annual report of the Chief Medical Officer, Ministry of Health for 1935, an outbreak occurred in Lancashire in which beef from the carcass of an animal suffering from Salmonella septicæmia when slaughtered, was responsible for 174 cases with 8 deaths. The carcass was not noticeably unhealthy, but from all parts of it examined S. typhi murium (aertrycke) was isolated. In many of the cases the meat had been consumed in the form of pressed beef.

In a number of outbreaks due to the consumption of meat from a diseased animal (especially in Germany), it has been ascertained afterwards to have been 'emergency-slaughtered.' According to German authorities, four-fifths of the outbreaks of meat poisoning are due to cattle slaughtered when on the point of death, suffering from some septic or diarrhœal condition, slaughter having been effected under private or subsequently unascertainable conditions. In 61 large outbreaks of food poisoning between 1869 and 1898,

affecting 5000 persons with 76 deaths, the meat of cows was incriminated in 38, of calves 15, of oxen 3, of pigs 2 and of horses 2. Meyer (1929) recorded 120 outbreaks between the years 1923 and 1928 which were due to this cause.

Doubtless under inadequately controlled conditions, opportunities may occur for the transference of infection from diseased animals to healthy meat where the emergency slaughter of sick animals takes place in the same room as healthy carcasses are being dressed. The careless handling of local lesions of a diseased animal may also be a possible source of Salmonella infection. Ostertag has pointed out that as a result of the analysis of 85 recorded outbreaks of food poisoning during the years 1880–1900, most of which occurred in Germany, " prove anew the especially dangerous character of the meat of calves affected with sepsis in association with umbilical affections and also of cows which have to be subjected to emergency-slaughter on account of inflammatory processes after parturition or on account of peculiar affections of the intestines and udder."

Savage (1920) investigated and recorded the following interesting and typical outbreak definitely connected with food from a diseased animal :

" On Friday, 8th May, 1908, in Murrow, a village in Cambridgeshire, a woman purchased some pork bones from a local butcher and that evening used them to make some brawn. The following morning the brawn was emptied out of the saucepan in which it had been made and, without cleansing the vessel, potatoes and asparagus were cooked in it. These vegetables were eaten for midday dinner by 4 persons, and all were subsequently attacked with vomiting, diarrhœa, and the other symptoms of food poisoning, two in the night and two next morning. The husband who was away at midday remained well and unaffected.

" On the Monday, two days later, the brawn made up into pork cheeses (a local name for brawn) was given away to three different neighbours and was consumed by a further 14 persons, all of whom were attacked with similar symptoms, after an incubation period varying from 12 to 48 hours. Three of the 18 attacked died. No one eating the brawn escaped.

" None of the brawn was available for examination, but from the only fatal case investigated a Gaertner group bacillus (B. aertrycke) was isolated, and its connection with the outbreak was further proved by the fact that it was agglutinated in high dilution by the serum of three survivors.

" The brawn was home prepared, and the materials were slowly heated for several hours with a short boil at the finish, but obviously actual boiling temperature was not reached. That the Gaertner bacilli were present before preparation and survived cooking is evident from the infection imparted to the vegetables through the uncleansed saucepan. Further inquiries elicited that the pig which supplied the bones for the brawn had suffered from local injury or disease of one leg, no doubt due to infection by this food-poisoning bacillus."

B. aertrycke and B. enteritidis have been isolated in recent years from sick animals by several observers, including Gheorghiu and Costin (1927) ; Lachenschmid (1931) ; Edwards (1934) ; Lovell and Hughes (1935) ; Hohn and Herrmann (1935) ; and Ferrario (1935).

With regard to sources of infection (reservoirs of Salmonella bacilli) outside the animal body, Savage and Bruce White (1925) say : " This is possible, but we have no facts which suggest that it is probable. These bacilli are not natural intestinal inhabitants, and therefore it is not to be expected that they will be present in manure or other form of animal excreta unless derived from an animal infected with a Salmonella strain. They have a resistance comparable to that of the typhoid bacillus, and extended work with that organism has shown that its life is measured by days under purely saprophytic surroundings. Our few examinations of manure, dust, and other suspected material has always yielded negative results, and at present we have no data to support this hypothesis."

## B. Milk from an Infected Animal

There are a number of recorded outbreaks where Salmonella bacilli have been isolated from the milk of a diseased cow and from the fæces, i.e. Kinloch, Smith and Taylor (1926), McAllan and Howie (1931), Savage (1932).

In the annual report of the Chief Medical Officer, Ministry of Health for 1936, a large milk outbreak (130 cases), which was investigated by Conybeare and Thornton, occurred in a town in Wiltshire, chiefly among school children. Salmonella (B. enteritidis Dublin) was isolated from both the milk and from the fæces of one of the cows. According to the report, " it is comparatively rare for the mode of transference from the animal reservoir to the human patient to be so clearly established. The Dublin type has

long been known as specially affecting bovines, chiefly in the form of epizootics among calves."

### C. Infection Transmitted by Duck Eggs

The infection of ducks and their eggs by Salmonella strains, including B. aertrycke and B. enteritidis, is not uncommon. The importance of the duck's egg as conveying Salmonella infection was brought into prominence by Scott (1930, 1932, 1933), Clarenburg and Dornickx (1932), Lovell (1932), Seligmann (1935). Hohn and Hermann (1935), Jansen (1936). Scott (1933) suggested that the eggs are probably infected during their formation in the oviduct, but the bacilli may gain access through the intact shell. In his paper on the subject (1930),he mentions several outbreaks, including a typical one which occurred at Darlington in 1927. A trifle was consumed by 10 out of a party of 12 persons. All the 10 were seriously ill, owing to an aertrycke infection, while the two who had no trifle but had shared in all the other food, remained well. The cream of the trifle had been prepared by whipping the whites of ducks' eggs.

Beller and Reinhard (1934), who examined 1500 ducks' eggs from 34 farms in Germany, found that about 1 per cent. contained Salmonella organisms.

The Chief Medical Officer of the Ministry of Health, in his annual reports for 1926, 1929, 1933 and 1938, drew attention to the strong circumstantial evidence incriminating insufficiently cooked ducks' eggs as the cause of severe and fatal food poisoning, and the possibility that many cases of gastro-enteritis in which the hypothesis of alimentary infection appears impossible, since the single sufferer has consumed only food and drink shared with impunity by others, may be explained by the ingestion of a Salmonella-infected egg. " Fried, lightly boiled, in creams, custards or mayonnaise, and most of all in the raw form, as egg-flips, etc., ducks' eggs are capable at all seasons of the year of producing severe gastro-enteritis and fatal septicæmia."

### D. Infection Transmitted by Rats and Mice

Rats and mice are known to be susceptible to infection by Salmonella. The rat is probably the host of B. enteritidis and the mouse harbours B. typhi-murium, and they may, when so infected or in the carrier state, excrete the organisms in their fæces and urine for considerable periods. Food prepared and left exposed

in insanitary premises attracts rats and mice and is liable to be so infected.

Savage and Read (1913) examined the internal organs and intestinal contents of 41 rats and were able to isolate Salmonella enteritidis from the spleen of 5 rats. No member of this group was isolated from the intestinal content. This proved that while rats may be infected with organisms of the Salmonella group, these bacilli are not normal inhabitants of their intestinal tracts.

Savage and Bruce White (1923) examined 96 rats caught in two slaughter-houses. They isolated B. enteritidis from 6 of them; 3 of them harboured the organism in their intestines, thus demonstrating that the infection of meat from this source is possible. Meyer and Matsumura (1927) made an examination of 775 wild rats from the district of San Francisco and found 58 harboured B. enteritidis (28) or B. typhi-murium (30).

The annual report of the Chief Medical Officer of the Ministry of Health for 1936 records an investigation of the Salmonella infections of rats by Khalil, which " illustrates the importance of the animal reservoir in which the various Salmonella types maintain their existence :

" LIVERPOOL RATS, 1936

| Rats Examined. | Number Positive. | Percentage Positive. | Rats Examined. | Number Positive. | Percentage Positive. | Rats Examined. | Number Positive. | Percentage Positive. |
|---|---|---|---|---|---|---|---|---|
| 250 | 44 | 17·6 | 250 | 10 | 4·0 | 250 | — | 0·4 |
| Jan. to Mar. | 26 aertrycke | | Apl. to June | 1 aertrycke | | July to Aug. | 1 enteritidis | |
| 175 City | 16 enteritidis | | 175 City | 7 enteritidis | | 175 City | | |
| 75 Port | 2 Newport | | 75 Port | 1 Newport | | 75 Port | | |
| | | | | 1 Thompson | | | | |

" It will be seen that several of the types common in food poisoning can be found in the rat in much the same order of

frequency ; the enteritidis type (Gaertner), however, appears more commonly in the rat than it does in man, and there is reason to believe that it is the rat type in particular, just as the Dublin type is bovine. As regards the other types, individual rats may become infected by them, probably by eating infected food, but, except in the case of aertrycke infection, rat epizootics apparently do not result from them."

As regards outbreaks, Jones and Wright (1936) record the case of a child who died from eating infected dried milk which was kept in an uncovered container. Mouse fæces were found in the food, and from these, as well as from the powder, B. typhi-murium was isolated. From the intestines of mice caught in the house within the next few days the same organism was cultivated.

Jordan (1929) stated that the widespread distribution of Salmonella enteritidis in rats and mice might be of considerable epidemological importance. He reported that rats caught in various parts of Chicago frequently yielded this organism.

Savage (1932) says : " The fact that specifically infected rats and mice are vehicles of infection in food poisoning may be taken as established, and the infrequency of actual proof is probably due to the difficulties of the quest and a good deal to the failure of those responsible for collecting material, etc., to grasp that importance of this line of enquiry."

Cultures of bacteria are sold frequently for the extermination of rats and mice. The Chief Medical Officer of the Ministry of Health, in his annual report for 1932, issued a warning regarding the use of virus preparations. He said : " In 1929, and again in 1931, I called attention to the danger to human beings involved in the use of ' virus ' preparations for the destruction of rodents and to the great caution necessary in employing them in circumstances in which contamination of food or drink might occur, either with the virus material itself or by the excreta of rats and mice infected with it. The occasion for this repetition was the outbreak of 1st November, 1931, in which 38 cases of food poisoning and 1 death was traced to the use of such a virus in a bake-house in Wigan.

" The vehicle was stuffed cow's heart or stuffed roast pork, the stuffing being the only part actually infected, and 2 patients consuming the stuffing alone. The stuffing when first made was not infected, as portions taken at once to a branch shop and part sold on 3rd November were harmless. The part which became infected remained on the shop counter overnight in a glass vessel placed on a bench in a passage at the rear of the shop. Next day it was

mixed with the rest of the stuffing and this was poisonous in every case. On 26th October a mouse destruction material had been used on the premises. From remains of a tin of this bacterial material, from mice trapped on the premises, from the organs of the patient who died, and from the excreta of other sufferers Dr. Scott isolated bacilli which were identical in every particular with one another and with B. enteritidis " (Savage, 1932).

A number of outbreaks have been recorded in which infection has been traced to the use of virus preparations for the extermination of rats and mice by Tanner (1933), Jordan (1930), Boeker and Kauffman (1930).

## E. Human Carriers

Human beings infected from animal sources may, during illness and for some limited period after, discharge pathogenic bacilli in their fæces and contaminate food by handling. This problem is a difficult one, and proved cases of infection from human sources are very few. There is little evidence that Salmonella organisms are able to live and multiply in the human intestine, and they usually disappear soon after recovery from the illness. Instances, however, have been recorded where B. aertrycke has been found in the fæces of persons not suffering from food infection. Temporary carriers may be more common than is generally supposed.

In 1932 Savage expressed the view that the human carrier plays quite an insignificant part in the causation of food poisoning.

Among food-poisoning outbreaks ascribed to human carriers was one which occurred in France in 1917, involving some 1060 cases. It was investigated by Perry and Tidy (1919) and was proved to be due to B. aertrycke. One case remained positive for 14 weeks. In America, however, Jordan (1917) and Geiger (1923) attach importance to this source of infection.

Topley and Wilson (1936) remark : " That chronic Salmonella carriers, analogous to chronic typhoid carriers, are uncommon, there seems to be little doubt ; but considering the frequency of rodent typhoid, it would be surprising if sporadic infections of human beings, particularly those used to handling food, did not occur fairly often. . . . Temporary carriers of this type must always be a danger to the human population. Their detection by bacteriological methods is bound to be difficult, since their carrier condition will often have cleared up before suspicion is cast upon them."

## F. Flies as Carriers of Infection

With regard to flies being possible vehicles of Salmonella infection, it is well known that these insects assist in spreading many dangerous diseases, and that the organisms may be conveyed on their legs, wings and bodies, or in their crops or intestines, but there appears to be no evidence that flies naturally harbour Salmonella bacilli.

Graham Smith (1914) found that with flies infected with B. enteritidis, this organism can be found in the contents of their crops and intestines at least seven days after infection. A comprehensive study of bacteria on flies was recorded by Scott in America (1917). He found a seasonal variation in the bacterial content of flies in Washington, and that the greatest number of bacteria was found in the summer months (see also Nichol, 1917, Bishopp and Laake, 1921). Graham and his colleagues (1922) indicated that flies can transport type A toxin of Cl. botulinum on their feet and bodies, and also that it may be regurgitated from the crop material.

A number of interesting experiments were recently carried out in America by Ostrolenk and Welch (1942) on "The House-Fly as a vector of food poisoning organisms in food-producing establishments." These were summed up by Savage (1942) as follows: "Specially reared flies were fed with infected food containing Bact. enteritidis, steps being taken to reduce surface contamination to a minimum. Not only were the flies readily infected but in transference experiments they transmitted the organism to fresh flies and these again to further series of flies. The organism was invariably isolated from the intestinal tracts and also from fly drinking water, food and the surfaces of the cages.

"Longevity experiments demonstrated that this organism survived for at least 20 days and, if they had been carried further, probably for the entire duration of the life of the fly (4 weeks). Fly eggs planted in mash infected with Bact. enteritidis resulted in infected maggots, pupæ and adults. Infected flies given access to healthy mice resulted in the infection of a number of the mice with some deaths. Further experiments also demonstrated that the transfer of the Salmonella from the infected mice to healthy flies was possible and took place in a number of instances."

Although the seasonal prevalence of these insects and that of food-poisoning outbreaks are somewhat similar, the isolated nature of the outbreaks does not seem to favour fly-borne infection.

Savage (1941) remarks: "There is slightly more probability

that they might convey dysentery bacilli responsible for a food-poisoning outbreak, given all the favourable conditions."

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CHAPTER VII

# PREVENTION AND CONTROL OF BACTERIAL FOOD POISONING

THIS is a problem. In discussing any hygienic and other preventive measures, several important relevant matters require special consideration.

It has been often stated "that to control infectious disease knowledge of its occurrence is the first line of defence."

Up to the end of 1939 food poisoning was not a notifiable disease, except in the County of London (Public Health (London) Act 1936). In consequence, no reliable figures outside this area were available, and although from time to time the Ministry of Health issued valuable informative memoranda and advice dealing with the subject, many outbreaks and individual cases of food infection and intoxication occurred in the towns and rural districts which were never brought to light and consequently never investigated; moreover, unless each reported outbreak is systematically and scientifically studied, the information and data obtained often prove to be of little value.

This unsatisfactory state of affairs should be remedied by the introduction of notification under the Food and Drugs Act, 1938, which came into operation in October, 1939.

Although primarily a consolidating Act, it contains many valuable amendments of previously existing laws, especially regarding the precautions necessary against the contamination of food.

## Compulsory Notification of Cases of Food Poisoning

Under Section 17. " If a registered medical practitioner becomes aware, or suspects, that a patient whom he is attending within the district of any local authority is suffering from food poisoning, he shall forthwith send to the Medical Officer of Health of that district a certificate stating (*a*) the name, age and sex of the patient, and the address of the premises where the patient is, and particulars of the food poisoning from which he is, or is suspected to be, suffering, and also stating whether the case occurs in the private practice of the practitioner, or in his practice as medical officer of a public body or institution." (There is no obligation on householders and parents.)

Under Section 18, " Provision as to suspected food. If the Medical Officer of Health of a district has reasonable ground for suspecting that any food of which he, or any other officer of the local authority of the district, has procured a sample under the provisions of this Act is likely to cause food poisoning, he may give notice to the person in charge of the food, that until his investigations are completed, the food, or any specified portion thereof, is not to be used for human consumption and either is not to be removed, or is not to be removed except to some place specified in the notice.

" A person who uses or removes any food in contravention of the requirements of a notice given under this subsection shall be liable to a fine not exceeding ten pounds.

" If, as a result of his investigations, the Medical Officer is satisfied that the food in question, or any portion thereof, is likely to cause food poisoning, he may deal with it as food falling within subsection (1) of Section 10 of this Act (Examination of food and seizure of unsound food), and subsections (2) and (3) of that section shall apply accordingly, but if he is satisfied that it may safely be used for human consumption he shall forthwith withdraw his notice."

This compulsory notification will not only result in all cases of food poisoning being studied on the spot at the earliest possible stage, and comprehensive investigations made into the suspected sources and modes of infection, but will enable the detailed results of the investigations and the confirmatory evidence of the bacteriological and pathological findings to be recorded, analysed and classified. In this way much light will be thrown on unsolved problems of food-poisoning outbreaks and a vast amount of valuable and definite information gained. As time goes on, it should be possible to ascertain from this accumulated knowledge exactly how food becomes infected by members of the Salmonella group and to discover with certainty the reservoirs, habitats and paths of infection. This would allow of such exacting preventive measures being instituted as would eradicate food-poisoning outbreaks, or at least reduce their incidence to a minimum. It has been suggested that coroners should be asked to report to the Medical Officer of Health of the district concerned, deaths of all persons upon whom inquests are held where the cause of death is associated with some form of food poisoning.

In the light of present knowledge, hygienic and other measures of prevention can only be adopted as will tend to control to some

extent, likely sources of infection. It must be remembered that the prevention of food-poisoning outbreaks is dependent upon the application of bacteriological knowledge of the organisms concerned to the practical problems of food distribution and food handling.

"As soon as an animal is killed, questions of hygiene, as they are commonly understood, become of paramount importance. Micro-organisms must be prevented from contaminating the carcase as far as possible. So far, however, no one has succeeded in devising an aseptic method of slaughter. Micro-organisms do obtain access to our meat, and all we can do is to devise means of decreasing their numbers and arresting their growth.

"Contamination of meat is greatest on the slaughter floor, and it is at this point in particular that rigorous hygienic precautions are essential in order to produce meat with the maximum keeping qualities. The primary sources of infection are chiefly the feet, hides or skin, and the intestines of the animal. The infection is transmitted to the carcass by the hands, knives, swabs, washing water and clothing of the operatives, and, in fact, it is reasonably well established that by comparison air-borne infection is negligible.

"Control of temperature is the most important weapon that we have to reduce or prevent the growth of micro-organisms on meat. There are, however, other agents, including salting, smoking and drying, gaseous inhibitors such as carbon dioxide or ozone and ultra-violet light" (Callow and Morgan, 1938).

## Legislation

The question arises, what are the existing legal measures for the general control of meat and meat foods which should prevent diseased, infected or unsound meat reaching the consumer ?

The Public Health (Meat) Regulations, 1924, together with the instructions on a "System of Meat Inspection," issued by the Ministry of Health (Memo. No. 62, Foods, 1922) constitute a valuable safeguard against the conversion of diseased or unsound animals into human food.

Among the most useful provisions of the Public Health (Meat) Regulations are Sections 8 (2) and 10. Section 8 (2) requires notice to be given of the times of slaughter of animals, so that necessary inspection of the carcasses and offal can be made ; also when sick or injured animals are to be or have been slaughtered for emergency reasons, the carcasses of which are for human con-

sumption. In this connection it would be preferable, however, if the slaughter of sick or injured animals was excluded from private slaughter-houses because of the grave risk of transference of infection from sick animals to healthy meat.

Topley and Wilson (1936) remark : " The hygienic precautions necessary to prevent food poisoning concern the whole course of the food from the slaughter of the animal to the final preparation for consumption. A thorough system of meat inspection is essential. The meat of animals that are ill or are emergency-slaughtered should, as a rule, be condemned. To this precaution alone Meyer (1916) attributes the comparative infrequency of meat poisoning in California, where it is known that calves are infected with B. enteritidis."

In Section 10 of the Act, " The person by or on whose behalf an animal is slaughtered for sale for human consumption shall not cause or permit the carcass of the animal, including the mesentery and internal organs other than the stomach, intestines and bladder, to be removed from the place of slaughter until such carcass with its organs has been inspected, or its removal has been authorised by an Inspector of the Local Authority."

## Knackers' Yards and Private Slaughter-houses

Section 19 of the Food and Drugs Act, 1938, deals with meat from knackers' yards. " No person shall sell, or offer for sale, for human consumption any part of an animal which has been slaughtered in a knacker's yard."

The supervision of privately-owned slaughter-houses is difficult even in well-organised centres, but the occupation of such buildings by unscrupulous dealers in a remote district is a menace to public health, more especially where the business of butcher and knacker is combined. There is no doubt that a certain amount of traffic in doubtful carcasses still goes on.

Martin (1940), referring to this important subject, remarks : " This is about as far as legislation can go, but there are many loopholes, and considerable vigilance will still be necessary to prevent this illicit trade, and with animals killed at any odd time of the day or night, the meat taken into towns by fast motor vehicles and quickly disposed of, it is not going to be easy. The provision of clearing-houses to all towns, to which all meat coming into the district from outside must be sent for inspection before exposure for sale in the town, and a universal system of marking, would of course solve the problem."

## Public Slaughter-houses

Section 60 of the Food and Drugs Act, 1938, gives power to the local authority to provide public slaughter-houses.

The abolition of private slaughter-houses would result in centralised slaughter in modern, well-organised abattoirs, where an efficient ante-mortem and post-mortem inspection would be carried out by a trained technical staff having a sound clinical knowledge and experience of the diseases of animals, and thus prevent the release of diseased or unsound meat for sale and consumption.

It is essential that slaughtering and inspection of food animals should be undertaken in the same place.

Fourie (1936), referring to clinically sick animals remarks: "There should be no great difficulty in recognising many of the cases which could be responsible for food poisoning. The ante-mortem inspection for the recognition of such cases is of fundamental importance, as in many cases the naked eye appearance of the carcass and the lesions in the organs are such that they are not easily recognised. These are the dangerous cases, as the organisms may actually be present in the musculature, and under conditions of summer temperature may multiply very rapidly and produce their toxins in the meat."

Incidentally, the better provision for slaughtering and cooling meat and diminished handling would favour its presentable appearance when exposed for sale. The consumer would have a guarantee that home-killed meat was good and wholesome.

Bacteriological examination of suspected flesh, organs and glands of food animals should, where possible, be carried out, although this seems hardly practicable as a general preventive measure. On the other hand, a bacteriological laboratory is an essential part of an up-to-date abattoir. In spite of all precautions at the time of slaughtering, meat infected with Salmonella organisms occasionally passes the first line of defence and finds its way on to the market.

The ante-mortem examination of food animals is covered by the Diseases of Animals Act, 1894, and the Tuberculosis Order of 1938.

## Centralised Slaughter under Government Control

Shortly after the outbreak of the present war, the Government decided to centralise and control the slaughter of all animals destined for food.

This centralisation will greatly facilitate inspection of animals before and after slaughter, particularly in small towns and rural districts, where private slaughter-houses are situated some distance apart. It should also prevent a leakage of unsound meat and improve matters generally from a public health point of view.

In October 1940 the Livestock (Restriction on Slaughtering) No. 2 Order was issued by the Ministry of Food.

## Supervision of Meat Foods

Statistics reveal that meat and 'made-up' meat foods are amongst the principal articles which act as vehicles of Salmonella infection and intoxication. It is well known that the surfaces of flesh foods have a high water content and are subject to bacterial activity, especially during exposure in slaughter-houses, shops, markets, stores, transportation, etc., because of inadequate protection or refrigeration. Thus they furnish suitable media not only for the growth of non-pathogenic organisms, but for certain members of the Salmonella group of bacilli. Research has shown that actual penetration of the bacilli below the surface, i.e. between the muscle fibres, deep into the meat, ordinarily takes some time to accomplish but is influenced by three factors—the temperature at which the meat is maintained, the amount of handling and the time interval.

Savage and Bruce White (1925) in their study of 100 recent outbreaks of food poisoning state : " There are at least four reasons which justify and emphasise the need for special control. These are :

" (*a*) They are foods made from pieces of meat, and therefore the chances of tracing the animal from which derived are limited. The great help afforded by an examination of the viscera of the animals supplying the meat is wanting.

" (*b*) They are foods which are subjected to considerable manipulation and therefore are especially liable to bacterial contamination.

" (*c*) They are mostly foods which are heated and then subjected to slow cooling, a procedure which facilitates and promotes bacterial growth in what is a suitable nutrient medium.

" (*d*) They are varieties of foods the preparation of which is often carried out as an adjunct to other businesses, such as slaughter-house work either on the same or adjacent premises, which facilitate specific infection.

## Food Poisoning

"The 'made-up' foods here indicated include such foods as brawn, potted meat, meat pies, sausages. It is somewhat difficult to frame an inclusive definition.

"The records of food-poisoning outbreaks show how important is this class of vehicle as a source of outbreaks, and how frequently their manufacture is associated with conditions which facilitate bacterial infection."

The Chief Medical Officer of the Ministry of Health in his annual report for 1937 says: "The risk in food factories producing large quantities of meat preparations (brawns, sausages, etc.) that an infected pig's carcass might spread infection to other foods in the process of manufacture was well illustrated in a widespread outbreak of food poisoning in the West of England in May and June 1937. No less than 148 cases of rather severe gastro-enteritis and 4 deaths due to infection with Salmonella enteritidis were traced to the consumption of various products (pressed beef, galantine, bath chaps, etc.) from a single factory. The outbreak was investigated by Dr. Conybeare, who concluded that the accidental admission of a Salmonella-infected pig carcass to the factory was the primary event."

In this connection it is of interest to mention the following recorded outbreak which occurred in the Exeter district in 1938, and involved some 50 persons. The outbreak was traced to brawn prepared on premises where calves which were recovering from calf dysentery were housed. The causative organism (S. Dublin) was isolated from the brawn and from numerous sufferers.

This organism is a common cause of infectious diarrhœa in calves and can be conveyed to man by cow's milk.

## Registration of Premises

Premises used for the manufacture or sale of ice-cream or preserved food, etc., are dealt with in the Food and Drugs Act, 1938. Section 14, as follows: "No premises shall be used for the sale, or the manufacture for the purpose of sale, of ice-cream, or the storage of ice-cream intended for sale; or the preparation or manufacture of sausages or potted, pressed, pickled or preserved food intended for sale, unless they are registered under this section for that purpose by the local authority and a person who uses any premises in contravention of the provisions of this subsection shall be guilty of an offence. For the purposes of this subsection, the preparation of meat or fish by any process of cooking shall be deemed to be the preservation thereof. . . . This section shall

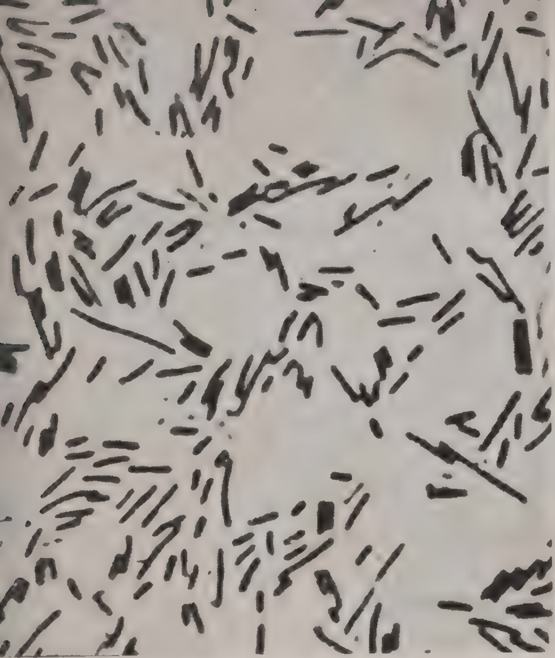

FIG. 11.—Bacillus proteus vulgaris.

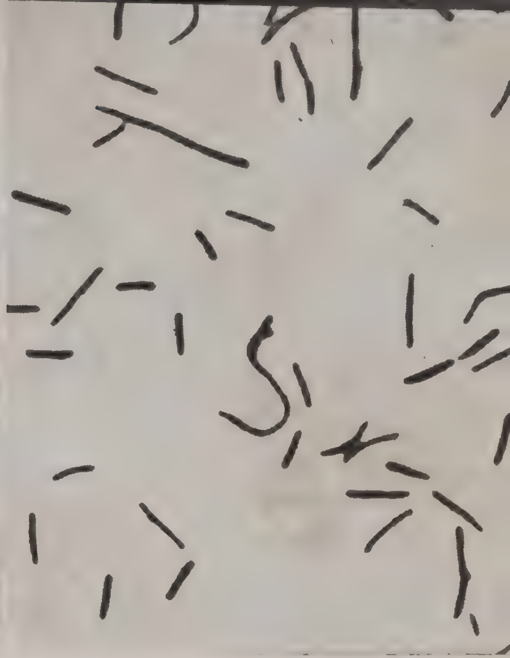

FIG. 12. Bacillus enteritidis (Gaertner

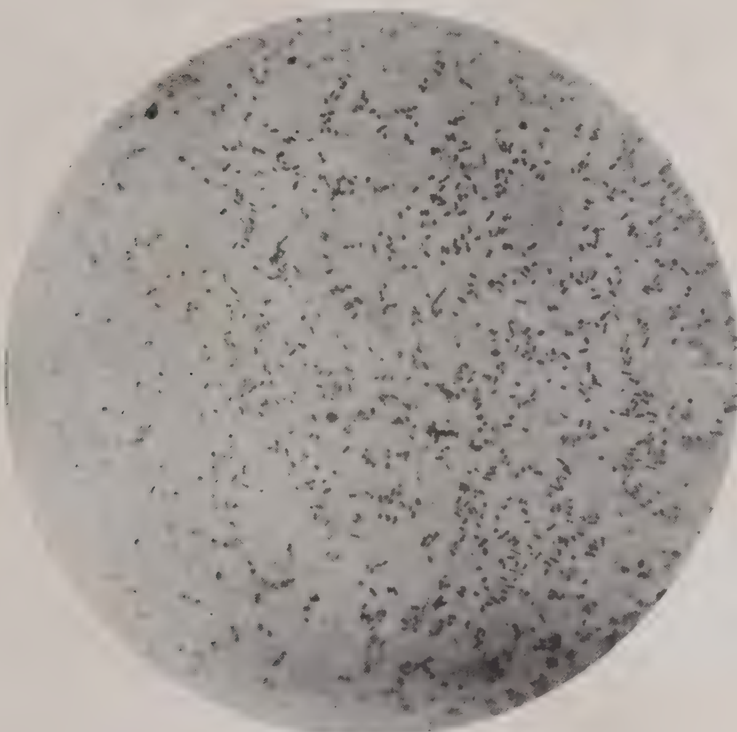

FIG. 13.—Bacillus aertryche × 1000 diam.

*Courtesy of the Liverpool Co-operative Soc*

FIG. 15. Bread Wrapping Machine.

FIG. 16.—A section of the food research laboratories of the British Food Manufacturers' Research Association.

not apply in relation to premises used primarily as a club, hotel, inn or restaurant, and in relation to premises used as a theatre, cinematograph theatre, music hall or concert hall."

Section 13 of the Act deals with the precautions against contamination of food, i.e. provision as to rooms where food intended for sale is prepared or stored, etc.

The difficulties in tracing the original source of infection in food-poisoning outbreaks attributable to meat are obvious. During the various processes of handling and distribution, opportunities for contamination are many. Further difficulties arise in assigning a particular piece of meat to a specific carcass after it has been passed through a large retail or manufacturing establishment. Accurate records, therefore, should be kept, showing the original sources of the meat, viscera, etc., used in the manufacture of made-up food articles.

Strict attention to cleanliness in manufacture, preparation for sale (wrappings), storage and distribution of foodstuffs are of the greatest importance and would obviate many of the dangers of food acting as a vehicle of infection. These precautions apply especially to foods liable to imperfect cooking and where a period elapses before they are consumed.

## Handling and Wrapping of Foods

Section 15 of the Food and Drugs Act, 1938, provides for the making of byelaws by Local Authorities with respect to handling, wrapping of food, and the sale of food in the open air.

Cleanliness and personal hygiene of all food handlers are of paramount importance. Persons capable of transmitting infection, i.e. suffering from any derangement of the alimentary tract, must be precluded from the handling of food or food utensils. Mild cases of such illness must never be disregarded. It has been suggested that whilst the routine laboratory examination of all food handlers is not justified, either on financial or practical grounds, a modified system of laboratory and clinical control is worthy of trial.

## Human Carriers

Anent this, the Chief Medical Officer of the Ministry of Health (1937) remarks: "The protection of cooked foods from infection at the hands of the vendors in shops has been much discussed, and especially in America bacteriological control to exclude 'carriers' from this occupation has been suggested. It seems

plain, however, that any plan of this kind is impracticable, and that reliance should be placed rather on the provision of ample facilities for washing and on the inculcation of proper habits of personal hygiene among such persons. The increase in the number of shops selling cooked foods of all kinds, in urban districts especially, imposes on Medical Officers of Health the duty of careful supervision of their sanitary condition ; they are probably the chief source of the increased prevalence of the intestinal infections."

Stebbings (1940), New York State Department of Health, recently dealt with the general question of the examination of food handlers. He pointed out that it must involve a clinical history, physical examination, laboratory test and sometimes X-ray or other examination. He remarks : "This department is firmly convinced that, under the conditions in which it is possible to carry them out on a community basis, routine food handler examinations are unwarranted and are not to be considered a proper use for public funds."

There is no legislation which prevents the handling of meat by prospective purchasers—a practice still common in retail shops. Some retailers supply forks for the use of customers and have notices posted saying that the meat must not be handled.

## Preservatives in Food

The Public Health (Preservatives, etc., in Food) Regulations, 1925, amended in 1926 and 1927, prohibit the addition of preservatives to made-up foods, such as brawn, meat and potted food, etc.

## Cooking of Foods

The various processes of cooking, such as roasting, broiling, etc., may fail to kill all organisms in flesh foods, but they succeed in diminishing them considerably. Well-cooked food is less liable to cause food poisoning than raw or partly cooked food. Whatever danger there may be, either from infection or intoxication, is dependent upon storage with the incubation or re-infection of the cooked product. If the food is not eaten immediately after it has been cooked, it should be placed in a cool situation, refrigerator or ice-chest. The bacteria surviving in good, sound food, freshly and thoroughly cooked, are not ordinarily a menace to the consumer if such food is consumed at once.

Jordan (1931) remarks : "It must be remembered that in some outbreaks those persons consuming raw or partly cooked meat have

been affected, while at the same time others eating well-cooked meat from the same animal have remained exempt."

## Refrigeration

The use of refrigerators have many advantages in the storage of meat and other foods and provide a uniform temperature low enough to prevent the multiplication of bacteria whilst the food is actually in the refrigerator. Refrigerators, however, have certain limitations. It is not a steriliser and cannot render food safe if it has been infected by pathogenic organisms. These may multiply upon the meat or other food during the time it is out of the apparatus and be rendered temporarily inactive but not destroyed by refrigeration. They will rapidly multiply if the food again reaches a suitable temperature. The film of moisture caused by the 'thawing' of 'frosted' meat provides a suitable medium for bacterial activity. Meat may be stored overnight in a refrigerator and subsequently exposed for sale in a shop window, and if not all sold, will, perhaps, be returned to the refrigerator on a second or subsequent nights. This is a dangerous procedure.

## Prevention of Milk-borne Infections

This may be effectively accomplished by efficient pasteurisation, or some other adequate form of heat treatment, of the milk. After pasteurisation the cooling process is most important. In the preparation of ice cream, Buchan (1910) found an enormous increase of bacteria occurred during the process of slow-cooling employed after preliminary heating.

Cleanliness alone is not a safeguard against infection conveyed by the milk of a diseased cow, and such milk may even pass the routine bacteriological standards for cleanliness. This is referred to by the Chief Medical Officer of the Ministry of Health in his report for 1934 as follows: "Cleanliness, however, is important from an æsthetic and commercial standpoint. Dirty milk is not only æsthetically objectionable but it has also poor keeping qualities, and for this reason alone reputable firms are anxious to obtain their supplies as clean as possible. Whilst, therefore, cleanliness is desirable, cleanliness is not enough. Safety is the really important consideration, and in present circumstances the ordinary raw milk supply can never be regarded as safe. To ensure its safety, that is to say, its freedom from pathogenic organisms, suitable heat treatment such as that afforded by efficient pasteurisation is essential."

## Food Poisoning

In 1938 an amendment was made to para. 3 of Part III of the Third Schedule of the 1936 Milk and Dairies Order to permit licensing authorities to require milk pasteurising plants to be fitted with sufficient recording and indicating thermometers to ensure accurate control of processing.

## Paper Containers for Milk

Albeit these have been available for a considerable time, only in recent years has the dairy industry begun to use them, and this has instigated the development of methods to ensure their sanitary quality.

In the United States, where their use is rapidly increasing, the New York State Agricultural Experiment Station has been studying the sanitary condition of paper stock used for milk containers. Subjoined are their recommendations regarding the production and handling of these to prevent infection of the milk :

" 1. Use of virgin pulp only.

" 2. Pure process water and strict microbiological control of pulp and paper mills.

" 3. Suitable protection and wrapping of finished board.

" 4. Mechanical handling of board and containers at conversion factories and milk plants.

" 5. Protection of board, adhesives, moisture-proofing materials and finished containers, from careless exposure to human contact, contamination, dirt, flushing water or insects.

" 6. Detailed knowledge and careful selection of all materials composing the container, to avoid the possibility of incorporating substances having germicidal or bacteriostatic effects, the use of which is prohibited unless they have been shown to be non-toxic to human beings and without effect on milk."

The Geneva Conference, 1938, suggested that " Board prior to moisture-proofing shall not, at any time, exceed 500 colonies per gram of disintegrated board.

" The average bacterial content of finished containers should not exceed 50 colonies per container."

These standards are lenient, and workers in this field have shown that the average container on the market will meet them easily.

## Animal Vectors in Milk Outbreaks

It has been suggested that in outbreaks of food poisoning connected with milk and milk products, a possible animal vector

should not be overlooked, and that veterinary co-operation should be sought in order to secure a prompt investigation into the question of infection from a bovine source. The routine inspection of dairy cattle is now carried out by veterinary officers of the Ministry of Agriculture and Fisheries under the establishment of a national service of veterinary inspectors.

The Milk and Dairies Order of 1926 (amended in 1938) deals with infectious disease amongst milk handlers. The Public Health (Infectious Diseases) Regulations, 1927, empower the exclusion of a ‘ carrier ’ of typhoid or paratyphoid fever or dysentery.

## Ice-cream

Bacteriologists have stressed the danger of the infection of bulk ice-cream during dispensing. The factory-filled package has been recommended as a means of avoiding possible contamination. Paper cups, boxes, bags, etc., used for ice-cream, must be properly protected during storage and handling. All containers in factories should be assembled with as little handling as possible. There should be frequent sterilisation of plant and utensils in ice-cream factories.

‘ Aging ’ or slow cooling should take place in a separate room to that used for mixing and freezing. All ingredients used must be adequately protected against contamination during storage.

## Ducks’ Eggs

With regard to the prevention of illness from the consumption of infected ducks’ eggs (3 cases of which occurred in 1937), there appears to be no practicable method of preventing with certainty the occurrence of Salmonella infection in ducks, though their exclusion from access to human or animal excreta doubtless would diminish its frequency. Cooking the eggs thoroughly is the only real safeguard.

It is interesting to note that in Germany a law was passed which prescribed that all such eggs offered for sale must be indelibly stamped “ Ducks’ eggs. Boil.” All receptacles in which these eggs are kept for sale must bear the following notice : “ Ducks Eggs. To be boiled for at least 8 minutes or thoroughly baked.” In addition, the following notice must be displayed near ducks’ eggs where they are offered for sale : “ In order to prevent injury to health, ducks’ eggs should not be consumed raw or lightly cooked, nor used in the preparation of puddings, mayonnaise, scrambled eggs, fried eggs, pancakes, etc.”

The above preventive measures might be instituted with advantage in this country.

## Contamination of Food by Rats and Mice

Everything should be done to prevent the access of rats and mice to food destined for human consumption. This may be accomplished by the rat-proofing of buildings and stores and the storage of foods in rat-proof containers. Incidentally, food manufacturers, owners of warehouses and similar premises in which produce and supplies are subject to infestation by vermin, have found that the cost of proofing their buildings is in the long run the cheapest form of insurance, and it is without doubt the greatest factor in the prevention of infection of food by rats and mice.

Strict attention should be given to the storage and disposal of all refuse and garbage. Water tanks and cisterns must be provided with proper fitting covers. In warehouses, especially where dried food is stored, the water supply should be cut off ; this precaution often causes rats to leave the premises.

The measures for the destruction of rats and mice are well known and need not be described. Legislation : Rats and Mice (Destruction) Act, 1919 ; the Rats Orders, 1940 and 1941 ; the Food Control Committees (Destruction of Rats and Mice) Order, 1940.

## Rat Viruses

A word, however, may be usefully added regarding the use of rat viruses. Savage and Bruce White examined a selection of these preparations and confirmed the view expressed by others in the past that these strains (e.g. Danysz, Liverpool Virus, Ratin) are typical enteritidis forms.

Jordan (1931) states : " A real danger to public health undoubtedly resides in the employment of so-called ' rat viruses ' for the extermination of these vermin. . . . Its use in kitchens and pantries may be the direct cause of food poisoning. There are on record a score or more instances where the careless use of a commercial rat virus has been followed by human infection, sometimes with fatalities. Since the method has not proved of material value in the destruction of rodents, and is, moreover, open to the serious sanitary objection that the animals after apparent recovery may continue to carry *Salmonella* bacilli and so contaminate food, the employment of rat viruses seems without justification."

Leslie (1942) made some interesting investigations into the

principal viruses which are used for rat and mouse control in Great Britain, and gives a bacteriological classification of the cultures contained in these preparations.

His summary is as follows :

" 1. The six 'viruses,' Liverpool, Danysz, London, Ready Rat Relief, Institut Pasteur and Ratin, which are the principal bacterial cultures at present employed for anti-rodent control in Great Britain, have been examined.

" 2. By means of reciprocal absorption tests all these six strains were found to be serologically identical with *S. enteritidis* Gaertner, antigenic structure, IX : gom :

" 3. From the results of the fermentation tests, which may be used to subdivide this serological type, Liverpool, Danysz, Ready Rat Relief and Ratin were assigned to the var. Danysz subgroup ; while the London and Institut Pasteur strains could not be distinguished from the classic *S. enteritidis* type.

" 4. Both of these subgroups are pathogenic for man, and evidence is cited which shows quite clearly that human cases of gastro-enteritis have been caused by the use of virus preparations. There are, also, reasonable grounds for believing that these bacterial types may be pathogenic for a number of domestic animals, including some poultry."

## Canned Foods

During recent years, owing to the rapid improvements made in all departments of the canning industry, cases of poisoning to-day are caused far less by canned than by 'made-up' foods. Although as shown by statistics (1937) outbreaks due to toxic canned food do occasionally occur, the number of cases is infinitesimal when compared with the tremendous output of canned foods of every description which are consumed annually, practically in every civilised country.

Unquestionably the improvements in canning are due firstly to scientific research, and secondly to the general preventive measures adopted in the factories. These include the special supervision exercised both as regards the cleanliness of the employees, as well as the machinery, utensils, floors, walls and general fittings. The cutting up, mixing and cooking of the foods and filling of the cans are also carried out with as little delay as possible before sealing in order to guard against accidental contamination. As a result of continued research, the subsequent processing

methods have been largely standardised according to the contents of the cans.

Savage and Bruce White (1925) were of opinion that the outbreaks from the consumption of canned food were due to infection of the contents before the food was put into the cans and before they were hermetically sealed. They state : " The prevention of outbreaks from this cause is therefore in the main the problem of preventing infection before or during manufacture. Higher temperatures of ' processing ' play a part, but not a large part in view of the marked resistance of Salmonella group toxins to heat."

In food factories, accurate records should be kept of the original sources of all foodstuffs used in manufacture, so as to enable any suspected article to be traced. A system of coding, where the code marks could be disclosed to the central authority in the country of import, would materially facilitate investigation of any particular consignment.

Savage (1939) points out that " Canned foods share with all other foods the risk of being a vehicle to cause food poisoning. From a study of all the data, it can be definitely stated that canned foods are now considerably less liable than ordinary foods to be a source of food poisoning. This is conspicuously so for the more dangerous outbreaks associated with the presence of living bacilli. Liability to cause the milder outbreaks of toxin type still exists but is being reduced, and even for this type their incidence, bulk for bulk, is less than that of other foods."

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# PART II

## CHAPTER VIII

## CONTAMINATION OF FOODS BY POISONOUS METALS

CHEMICAL poisoning is comparatively rare in this country, but the salts of poisonous metals do occasionally find their way into foodstuffs.

Normally, vegetable and animal foods contain minute traces of many elements, and analyses have proved that at times such metals as copper, arsenic, iron, etc., are found therein, but usually only in very small amounts. Chapman and Lindon (1926) proved the presence of arsenic and lead in marine crustaceans and shellfish, and they came to the conclusion that the metals were derived from the sea water. Samples collected from the Thames and Medway each averaged about one-fortieth of a grain of arsenic per gallon (0·33 p.p.m.).

The eating of certain fish, such as plaice, which may contain this metal up to three parts per million, leads to the presence of quantities of arsenic in the urine within 24 hours ; but whether such forms of arsenic and lead are really poisonous to human beings is a matter for investigation.

The rôle which metals play in food is a complicated one. It is a well-known fact that they may combine with the protein, thus neutralising any poisoning effect and rendering the food more or less harmless, except where the metallic salts are present in excessive amounts. This probably explains the reason some individuals, exposed to metallic poisoning, show no symptoms and are able to ingest and eliminate quite large amounts of metal which may cause illness to others. Like all other kinds of food poisoning the susceptibility or idiosyncrasy of the individual is of importance.

Savage (1941) referring to investigations of chemical poisoning, remarks : " The history of the outbreak, the very rapid onset after consumption in the acute cases, and the characteristic metallic poisoning symptoms usually puts the investigator quickly on the track."

Owing to the widespread use of metals in the food industry, many manufactured products, during preparation, come into close

contact with machinery and containers during cooking, processing storage, transportation and distribution, and there appears to be little doubt from the work of analysts that a certain amount of metallic contamination does occur, the degree usually depending on the length of time the foodstuff remains in contact with the metal. As a result of modern chemical and biological researches manufacturers are now realising the great importance of the precautions that must be taken during the handling of all such apparatus, especially the limitations to be placed upon the storage of foodstuffs in contact with metals. Moreover, these researches enable selection of suitable equipment to be made for use in factories and other places where food is manufactured, prepared and stored. Tanks and other apparatus lined with glass, or other special materials, not subject to ordinary corrosion by the product which may be brought into contact with them, have been introduced, thus reducing metallic contamination to a minimum.

Increased public interest has affected the general attitude towards foods. Manufacturers appreciate the necessity for hygienic methods in preparing and handling foodstuffs and the importance of protecting them from contamination so that they may reach the consumer in the best possible condition.

## Canned Food

Formerly, cans were made by hand and solder was used for sealing the top, sides and bottom. Thus metallic contamination was likely to take place. In the manufacture of modern cans, however, no solder comes into contact with the contents ; it is only applied on the outside, the ends being put on by means of a metal-to-metal seam with a thin layer of rubber compound between, the effect of which is to prevent contact with any lead from the solder, so that the only contamination that can take place is from the tin and iron which is practically nil.

During the process of tinning it is not possible to obtain an absolutely perfect coating on the steel sheets (the amount of tin does not usually exceed 2 per cent.) consequently precautions have to be taken to prevent interaction between the containers and the contents. The cans are lacquered to obviate any corrosion and to avoid change of colour in the food. Results of investigations carried out at the Campden Research Station, however, proved that this small quantity of tin is removed during the first two months of storing.

Buchanan and Schryver (1908) in their report to the Local

Government Board, stated that " practically all foods canned in the ordinary way become to some extent contaminated with tin as a result of the contact of the food with the tin-plate of the can. Tin is taken up by meat extracts and essences to a greater extent than by most other meat foods. This results from the acidity naturally possessed by the meat extractives in these preparations. Certain canned fruits and vegetables, and foods such as canned soups, of which the latter form part, are also specially liable to take up tin from the can in consequence of their natural acidity. In such cases, tin may penetrate into the substance of solid foods, and in the case of canned foods, which consist of both liquid and solid portions, e.g. canned fruit, the solid portion may come to contain relatively larger proportions of tin than the liquid. This results from the fact that the tin, after solution in the liquid contents of the can, becomes in course of time absorbed to, or chemically combined with, the solid contents." In some canned meat and fish products, protection from contact with the can and subsequent discoloration is obviated by using paper liners.

Contamination of canned fruits and vegetables by tin has been thoroughly investigated at the Campden Research Station. The results of the experiments go to show that certain vegetables are liable to remove more tin from the surface of the container than do acid fruits, and it is recommended that the inner surface of the can be protected by a lacquer. Very encouraging results were obtained from cans in which the second coat of lacquer is sprayed on after the tins are made up, and thus any scratches in the first coat of lacquer which exposes the iron are covered by the second coat.

In 1934 it was recommended that one method for obtaining improved protection was to spray the interior of cans made from twice-lacquered plate with a third coat of a quick-stoving lacquer. Trials with English fruits, which normally give trouble, were carried out on these lines, with decidedly advantageous results.

In the Food Investigation Special Report No. 44, 1936, it is suggested that in all probability the corrosion of cans by foodstuffs will eventually be overcome by improvements in lacquers and methods of lacquering. Failing such a development, relief must be sought through improvements in the tin coating in the steel base, in the cold storage of canned goods and in the application of knowledge concerning the corrosion of tin-plate. The metals usually associated with the contamination of food are : arsenic, antimony, copper, lead, aluminium, tin and zinc.

Tanner (1933) states that metallic salts may reach foods in different ways, as follows :

" 1. They may reach the food by accidental mixing of the metal or its salts, as illustrated by contamination of sugar with arsenicals during shipment, preparation of foods in containers of unknown origin, etc.

" 2. They may reach the food by solution from utensils in which it has been handled or processed. Such agents which might add a deleterious metallic salt have, in the main, been eliminated.

" 3. They may be added to the food for some special purpose. The use of lead arsenate sprays for destroying insects on fruits and vegetables is a good example.

" 4. They may be naturally present. Some foods, such as marine products, contain appreciable contents of metals. Such metal is apparently bound with proteins in the food and is not available until released, to poison the tissue."

## Arsenic

Probably no metallic contamination of food is of so much interest or importance as that of arsenic. It is present in sea water and the soil, thus gaining access to both animal and vegetable products which go to make up the human diet. Arsenic often occurs as an impurity in many chemicals which are used in one way or another in the food industry, consequently it is easy to understand that contamination of food at times is liable to take place. Traces of the metal have been found in jams, sweets (Hutchinson, 1909–10), lemonade, liqueurs, sugar, marmalade (Rupp, 1908), treacle and syrups, some of which commodities are largely manufactured from glucose. The use of glucose as an admixture or an adulterant is open to serious objection, unless it is known to have been prepared with acid freed from any arsenical impurity. At one period it was common for sweets, etc., to be coloured with arsenical pigments, but under the Public Health (Preservatives, etc., in Food) Regulations of 1925, the use of metallic colouring matters and compounds of certain metals in food is forbidden.

Arsenic has been the cause of food poisoning on several occasions. One of the most notable outbreaks was in 1900 at Lancashire, Cheshire, and Staffordshire, where some 6000 persons were poisoned by the presence of arsenic in beer, 70 of the cases proving fatal (Reynolds, 1901).

The Commission appointed to investigate the outbreak recommended that the arsenic content of substances used in food

manufacture should not be greater than $\frac{1}{100}$th grain per lb. (=1·4 parts per million) for solids and $\frac{1}{100}$th grain per gallon for liquids, the arsenic being expressed in terms of the oxide $As_2O_3$. It may be mentioned in passing that Wynter Blyth states, " the smallest single dose of solid arsenic said to have proved fatal to a human being is ·16 grms. (2½ grains)."

## The Spraying of Fruits and Vegetables with Poisonous Insecticides

From time to time arsenic has been found in excessive amounts in the wrappings and skins of imported pears. In one case $\frac{2}{3}$rds of a grain per lb. was present in the wrappings and $\frac{1}{12}$th of a grain per lb. in the skins. In 1926 samples of apples were taken for analysis. Five of the samples of Jonathan apples imported from America were found to contain arsenic, in each case more than $\frac{1}{100}$th grain per lb. Samples of English apples were found to be free from the metal. Again the results of investigations on English, Canadian and American apples showed 11 of 24 samples to be free from arsenic, 9 contained traces and 4 appreciable amounts.

Tanner (1933) says : " In several quarters the widespread use of metals and their salts in the food industries is viewed with alarm. Myers and Throne (1929) have recently pointed out that the public at large is submitted to the same action of arsenic as are the insects on sprayed fruits. They pointed out that arsenic is passing into the circulatory system as evidenced by its secretion after eating food which contain it. They claimed that too little attention is given to arsenic in foods and that imperfect fruit would be preferable to contaminated fruit."

Suggestions have been put forward regarding methods for the removal of the poisonous residue on fruits caused by spraying. It has been found that brushing and wiping will not remove arsenical compounds, but by careful washing in solution of hydrochloric acid (strength from 0·125 to 2 per cent.) its removal can be effected. The fruit is afterwards washed in running water to prevent injury by the acid.

There are no regulations in this country dealing with the spraying of fruits and vegetables with poisonous insecticides. The United States of America, however, has devoted much attention to the subject and to the reduction of arsenic on fruit to less than 0·01 grain per lb.

The United States Bureau of Entomology and Plant Quarantine is carrying out a search for a substance which shall be as effective

as arsenical preparations without the disadvantages which the latter possess. ‘ Phenothiazine ’ is one substance which is said to give promising results. Experiments are being made to ascertain whether in practice it will prove a satisfactory and hygienically unobjectionable substitute for arsenical preparations.

## Antimony

The salts of this metal, which is widely distributed in nature and a powerful poison, are seldom a cause of food poisoning, but several outbreaks, however, have been recorded both at home and abroad, due to cheap enamelled-coated utensils which yielded up a sufficient quantity of antimony to cause serious illness. Three such outbreaks have occurred in this country, in Newcastle-on-Tyne (Dunn, 1928), Folkestone (Monier-Williams, 1929) and in London (Monier-Williams, 1932), and were caused by lemonade made from lemons or lemonade crystals which had been prepared or stored in enamelled jugs or pails. The citric or tartaric acid dissolved out dangerous quantities of antimony from the utensils causing illness in a large number of persons in each outbreak.

In the outbreak which occurred at a London hospital in 1932 during a nurses’ Christmas dinner, 65 persons were seized with acute vomiting, followed in some cases by collapse. The cause was found to be the lemonade which had been prepared from lemons in white enamelled jugs.

It would appear that during recent years antimony oxide being cheap has been widely used as an opacifying agent in place of tin oxide. Antimony pentoxide, while safe as an enamel ingredient, owing to its very slight solubility in acids, becomes reduced during enamelling to the poisonous trioxide which is easily soluble. The remedy is to have an enamel matrix insoluble in acids because of a sufficiency of silica which encloses the oxide particles.

The Ministry of Health (1933) issued a memorandum in which attention was drawn to the possible danger of antimony due to the use of enamelled vessels with acid drink, like lemonade. In 1934 a pamphlet (No. 73) by Monier-Williams on “ Antimony in Enamelled Hollow-ware ” was also issued by the Ministry of Health, in which he summed up the situation as follows : “ The recent outbreaks of poisoning are attributable to the presence of relatively large amounts of antimony trioxide in enamels which were relatively low in silica and therefore soluble. The enamel matrix was disintegrated by the acid and the antimony oxide dissolved. There is reason to believe that almost all enamels con-

taining antimony give up small amounts of the metal to food, and it is at least questionable whether the continued ingestion of these small amounts may not be injurious to health. It is suggested that total prohibition of antimony in hollow-ware might be found to be in the best interests both of the public and of the trade."

During 1934 an unusual case of contamination by antimony occurred in Japanese canned oranges. Some of the samples contained 0·14 grain of antimony per lb. (corresponding to 0·37 grain of tartar emetic). It was suggested, however, that the oranges became contaminated by being prepared in vessels coated with a soft antimony enamel.

The medicinal dose of a soluble antimonial salt should not exceed 1½ grains. A dose of 2 grains has proved fatal.

## Copper

Acute poisoning from the presence of copper salts in food seems to have been but rarely demonstrated, and there is no doubt that the toxicity of the metal has at times been much exaggerated. It appears to be difficult to detect ill-effects when copper is fed to experimental animals, and rats fed over a long period with food containing salts of the metals show no sign of chronic poisoning. The opinion has been put forward, however, that such experiments do not do away with the possibility of the metal having an irritating effect on certain organs of the human subject, as is known to be produced by retention of other heavy metals in the body. From some feeding experiments on healthy men copper was found to be retained in the liver, and it is inferred that such retention of the metal must be harmful.

Excessive amounts of copper in food would probably be avoided. For instance, its presence in condensed milk gives it a tallowy flavour and in ordinary milk an emery taste. At one period, salts of copper were added to foods, vegetables and condiments to improve the colour, but this objectionable and perhaps harmful addition is now forbidden by the Public Health (Preservatives, etc., in Food) Regulations, 1925. It is, however, still practised on the Continent, and in 1934 four samples of canned mixed vegetables imported from Belgium were found to contain 36 to 54 parts of copper per million.

For some years copper has been found present in imported concentrated tomato pulp (puree and paste). The main source of copper was found to be—

1. Traces of copper, normally present in the tomato.

2. Copper salts deposited on the tomato as a result of spray during growth containing the metal.

3. Copper derived from the copper vessels in which the pulp is concentrated.

At a Conference of Port Medical Officers in 1938, it was agreed that a limit of tolerance of 50 parts per million in the dried total solids would have to be complied with on and after 1st January 1940.

The amount of copper and other metals occurring naturally in foods is adequate to maintain the ionic eqilibrium of these elements necessary for their proper biological functioning. Clayton (1933) compiled the following table, showing the natural copper content of foods in parts per million :

| | | | |
|---|---|---|---|
| Eggs . | 1·1–1·19 | Beef liver . | 16·0 |
| White potatoes | 6·5 | Beef kidney . | 2·4 |
| Yellow ,, . | 4·1 | Beef tongue . | 1·2 |
| Black grapes . | 8·1 | Cow milk . | 0·5–0·85 |
| Navy beans . | 10·45 | Human ,, . | 0·5–0·6 |
| Lima ,, . | 8·6 | Butter . . | 0·6 |
| Yellow corn . | 16·6 | Cheese . . | 0·6 |

Grendel (1930) estimated that the daily diet of a child of 6 to 12 years old would contain 0·5–1·0 mgm. of copper and of an adult, 2·10 mgm.

The presence of copper salts in oysters is well known, especially those obtained from the Cornish beds, but a considerable number would have to be eaten to yield a poisonous dose of the metal. A few cases of poisoning supposed to have been caused by food cooked in copper vessels have been reported. Hebblewaite (1928) described an outbreak of illness which was traced to the use of large copper kettles, used in the preparation of foods for heating water. Another outbreak, reported by Gardner (1925), was caused by copper salts in bread which became contaminated by the copper machinery used in baking.

The tendency in modern food factories and other places where food is prepared is to replace copper equipment by other metals such as nickel, monel, and stainless steel, and even silver, particularly as copper is readily dissolved by acid or salted foods.

Copper sulphate has been used on occasions for preventing the growth of objectionable algæ in water supplies. Fowler (1905) stated that 2 pints of such water would contain $\frac{1}{4}$ grain copper. The dose of copper as an astringent is $\frac{1}{2}$ to 2 grains. He believed

that such water was harmless to consumers. Copper pipes are now in common use for the conveyance of water supplies in buildings.

## Lead

This is a familiar, dangerous and accumulative poison, and when taken into the human body in very small quantities over a long period of time, causes chronic illness which may terminate fatally. The metal, even in the form of its most insoluble compounds, such as the sulphate or carbonate, is affected by the gastric juices and may become absorbed into the system. The chromate and arsenate are the most poisonous salts of lead. Incidentally women are more susceptible than men to lead poisoning.

Poisoning by lead in food and beverages is mentioned in the works of Pliny, Hippocrates and many other philosophers and writers. In the 16th century, Eathius describes a type of colic which was associated with the drinking of certain wines. In 1757–67 it was discovered that such wines and ciders acted upon and dissolved the glazes of earthen vessels in which they were stored, the glaze of the vessels being compounded with lead oxide.

The possible effect of food or drink on the absorption of lead from the alimentary tract is a most important point and cannot be too strongly emphasised. Lead may be more rapidly and completely absorbed from liquids than from solids, which is dangerous and poisonous in the former, though not so in the latter. Experiments have established that milk interferes with the absorption of lead and is one of the antidotes prescribed for acute lead poisoning.

Every person absorbs minute quantities of lead, either through the alimentary tract from food or through the lungs from dust. It is accumulated in the teeth and bones of apparently healthy individuals in a comparatively innocuous form. Gusserow (1861) attributed this to the formation of a double salt of lead and calcium. Roche, Lynch, Slater and Osler (1934) found from 15 to 146 parts per million of lead in various bones from healthy subjects. The bones of individuals may contain 50 to 100 parts per million of the metal—equivalent to 0·75 to 1·5 grm. in the whole skeleton.

A normal person may excrete daily 0·05 mgm. of lead in the urine and 0·3 to 0·4 mgm. in the fæces ; the amount varies considerably in different individuals. Certain observers, however, are of opinion that the greater part of the metal ingested with food passes out unabsorbed in the fæces ; whilst others contend that the intestinal canal in addition to passing through unabsorbed lead,

acts, probably, as the most important agent for the excretion o[f] absorbed lead.

During the last few years investigations have proved that lea[d] is more widely distributed in food than is appreciated, althoug[h] the amount present in most foods is usually small.

Analyses have shown that lead has been found in natural foods such as fruits, vegetables, cereals and marine crustaceans, and tha[t] in certain foodstuffs it is sometimes present in appreciable amounts.

The British Pharmacopœia (1932) gives the dose of lead acetat[e] as ½ to 2 grains, equivalent to 0·3 to 1·1 grain of the metal.

A large number of different foodstuffs were examined in th[e] laboratory of the Ministry of Health (Monier-Williams, 1938). A considerable proportion of these contained no lead or less tha[n] 0·2 to 0·4 parts per million, but some were found to contain lead i[n] excess of 2 parts per million. The following is a selection fro[m] the list of articles so examined :

| Foodstuffs. | Lead in Parts per Million. |
|---|---|
| Peaches | 0·9 |
| Strawberries | 0·4 |
| Oranges (pulp) | 0·5 |
| Apples | 0·3 |
| Home-grown tomatoes | 0·4 |
| Canned peas (home-grown) | 0·8 |
| Green peas (fresh) | 0·2 |
| Rice | 0·4 |
| Self-raising flour | 2·4 |
| Milk chocolate | 1·2 |
| Sardine paste | 8·3 |
| Silds (in aluminium container) | 5·1 |
| Bloater paste | 0·9 |
| Meat extract cubes | 2·4 |
| Baking powder (alum and phosphate) | 7·1 |
| Indian tea (loose) | 10·2 |
| China tea (in lead foil) | 6·1 |
| Custard powder | 1·2 |
| Margarine | 0·3 |
| Blancmange powder | 1·0 |

Several relevant matters of importance in connection with the presence of lead in food require special consideration. Until recently the determination of very small quantities in food was extremely difficult, and methods for analysis could not be relied upon to give uniformly accurate results, especially when only minute traces of the metal were present. Monier-Williams (1938)

found that of the many possible combinations of published methods is one which includes extraction with ' dithizone ' and precipitation of the lead as sulphate and determination colorimetrically as sulphide. This method is capable of determining minute amounts (0·002 mgm.) of lead.

Of late years considerable controversy has arisen regarding the amount of lead in food which may be considered negligible from a health point of view.

It has been calculated that 2 mgm. of lead ($\frac{1}{32}$ of a grain) absorbed daily, undermines the constitution and may set up chronic poisoning with changes in the kidneys and arteries which shorten life. A daily intake of 1 mgm. or even less must be regarded with suspicion.

Analyses of the foods examined in the laboratory of the Ministry of Health, reveal that normally about 0·2 to 0·25 mgm. of lead is likely to be ingested daily with food and that the total intake from all sources would be 0·5 mgm. of the metal (1·22 mgm. in food, 0·20 mgm. in water and 0·08 mgm. inhaled as dust). If, however, certain items are added to the diet the total amount of lead ingested would become excessive.

While the lead content in the majority of foods is very small, and its further reduction may be impossible, the metal may be present in some foods in considerable or even excessive amounts. The question arises whether it would be possible by the introduction of a standard (by specific legislation) either to eliminate the lead content in foods or reduce the amount to safe limits.

Monier-Williams (1938, " Lead in Food ") remarks : " The presence of lead in any particular food must be regarded, not only as a danger in itself, but as a contribution, more or less serious, to the total daily intake of lead from all sources. The aim should be to reduce the total amount of lead ingested in the diet to the lowest possible amount, and to this end every endeavour should be made to ensure that individual foods are prepared in such a way as to eliminate lead contamination as far as possible."

The Chief Medical Officer of the Ministry of Health (1938) in the Prefatory Note to the above pamphlet, sums up the matter as follows : " We are at present unable to say what quantity of lead may be considered negligible in food. It is, however, reasonable to infer that the harmful effects of continued small doses of lead begin from the moment the lead is absorbed and that the crude symptom-complex of chronic poisoning is merely the final stage of a long series of more subtle metabolic disturbances which elude our

imperfect methods of detection. In other words, the obviously harmful effect on the normal activities of the body of continued small doses of lead would seem to justify the assumption that there is no threshold below which still smaller doses can be regarded as being without some adverse effect. It would appear therefore that complete absence of lead from food is the ideal to be aimed at. For this reason it would seem inadvisable to set up standards by specific legislation which, by fixing permissible limits of contamination, would inevitably impede efforts to secure the reduction of lead in food to the lowest possible amounts. Our object must be to reduce the amount of any toxic substance in food to the smallest that can be achieved in practice, and this in many cases may be attained more effectively by administrative action than by the prescription of specific standards."

Food may be contaminated by lead in the following ways :

1. Exposure of food to dust containing lead as produced by the disintegration of lead pigments and paints during weathering.
2. The utilisation of solders, alloys, enamels and glazes containing lead, in the construction of receptacles, plant, machinery and apparatus, etc., which may come into close contact with food and food products, including certain beverages.

In a report to the Ministry of Health (Monier-Williams, 1925) on the " Solubility of Glazes and Enamels in Cooking Utensils," it was shown that food cooked in utensils having lead glazes might take up 3 to 4 parts of lead per million. This amount is increased if the food is allowed to remain in the vessels for any considerable length of time. He states : " The probability that undesirable constituents in insignificant amounts may be dissolved from enamelled hollow-ware during the ordinary processes of cooking may be regarded as remote."

Savage (1920) quotes an interesting case observed by Halenke and reported to Lehmann (1902). Two women ate cranberry tart for which they had cooked the cranberries in a cheap earthenware pot. Soon after eating part of the tart they became ill, one severely so. The glaze had been dissolved from inside the pot. A piece of the tart contained 160 mgm. of lead. It was estimated that each woman had consumed from 400 to 600 mgm. of malate of lead and that approximately as much as 100 mgm. had been dissolved in this single cooking. Cooking utensils now manufactured in Great Britain have a leadless glaze.

The metal is attacked by acids, alkaline foods and beverages.

and many instances of poisoning have been recorded as a result of such contamination both in this country and abroad.

Beer and cider are known to take up lead from vessels and pipes. In 1922, 93 cases with 1 death occurred in the County of Middlesex from the consumption of beer which had dissolved a substantial proportion of lead (up to 1·9 grains per gallon) from the enamel linings of the tanks in which it was stored.

Chronic lead poisoning occurred in a metropolitan borough in 1936. The beer had been drawn from the barrels through old lead piping. On analysis it showed the presence of 1 part per million of lead.

Recently an outbreak (Jackson and Jackson, 1932) of lead colic was reported from Devonshire. This was traced to cider drawn through tin-washed lead pipes connected to the casks and counter engines. Lead was present to the extent of $\frac{1}{10}$th to $\frac{1}{20}$th grain per gallon.

Samples of beer recently examined by local authorities showed from 0·3 to 3·0 and occasionally 9 and 13 parts per million of lead.

Bodron (1925) reported a curious epidemic of 37 cases of lead poisoning. This was traced to bread baked in an oven heated by wood derived from the breaking up of old boats, the wood being impregnated with paint containing lead salts. The vapour condensed on the loaves in the oven.

In modern public houses and hotels blocked tin pipes have taken the place of lead pipes. In many cases the lead pipes have been tin-washed, which affords little protection from the corrosive action of cider. Lead pipes lined with tin are not reliable unless the tin lining is thick and not damaged or worn. It has been suggested that pipes made of selected corrosion-resisting steel alloy for use with beer might be satisfactory.

The use of tin-washed and tin-lined pipes for beer and cider was discussed in the Annual Reports of the Chief Medical Officer to the Ministry of Health for the years 1932 and 1936 respectively.

The tin coating of tin-plate may contain small amounts of lead (less than 0·1 per cent. or even more in the case of commercial block tins), and it is possible that traces of lead find their way into the food.

In the Annual Report of the Chief Medical Officer of the Ministry of Health for 1935, attention is drawn to the importance of tea imported in lead-lined chests. Analyses of samples from these chests indicate that dry tea may contain considerable amounts of lead dust—varying from 10 to 20 parts per million.

Experiments have shown that about one-third of this lead goes into solution or suspension in the tea infusion as consumed. About 2 parts per million of lead in tea seems to be unavoidable as it becomes contaminated during the processes to which the leaf is subjected. The amount of lead in tea can be greatly reduced by using stout paper liners. Aluminium has been used with success in place of lead-lined chests.

Lead may gain access in small quantities to foil-wrapped articles, such as cheese and confectionery, but the use of paper interliners prevents contact between the food and the foil.

Trouble has been experienced of late years with imported sardines which were found to be contaminated with lead. Consignments representing thousands of tins of these fish were rejected at the Port of London. The subject was discussed in the Annual Report of the Chief Medical Officer of the Ministry of Health for 1936. In 50 samples examined by Local Authorities the lead content ranged from 10 to 80 parts per million.

Lampitt and Rooks (1933) gave the results of the examination of 596 samples, 30 per cent. of which contained from 10 to 90 parts of lead per million.

The contamination of the fish was derived from the grills (iron wire coated with solder containing a high proportion of lead) on which they were steamed. Steam condensing on the grills takes up the lead and contaminates the sardines.

At a Conference of Port Medical Officers of Health in 1933 it was agreed that sardines should be free from lead or contain negligible traces of the metal. As this would necessitate alterations to plant, etc., it was decided provisionally to take no action in cases where the lead content did not exceed 20 parts per million. As a result, a marked improvement took place, and at a second conference in 1937 the provisional limit was reduced to 5 parts per million for a limited period.

Ultimately sardines will be required to be free from lead or contain only negligible traces.

Lead has been present in smaller quantities in a number of other canned products, for example, tunny fish 13 parts per million, *pâté-de-foie gras* 10 parts per million, anchovies 8 parts per million, peeled shrimps 7 parts per million, crab paste 6 parts per million.

The Medical Officer of Health for the Port of London in his annual report for 1938 remarks : " Merchants have argued that the amount of lead in sardines is not dangerous to health, and have told me how many tins of sardines they have eaten, but when it is

pointed out to them that lead is an accumulative poison, that much damage may be done before definite symptoms of poisoning can be diagnosed, and that the trouble is not just the quantity ingested in sardines but the many small doses from the many different sources, they see our point of view and are anxious to know what steps can be taken to eliminate lead from their products."

3. The use of citric and tartaric acids, cream of tartar and acid calcium phosphate, synthetic dyes, etc., in the production of which materials containing lead have been used.

The contamination of citric and tartaric acids and acid calcium phosphate is mainly due to the use of lead utensils for concentrating and crystallising these chemicals. In 1907 (Local Government Reports of Inspector of Foods No. 2), at a special inquiry, the conclusion was reached that amounts of lead not exceeding 20 parts per million would not be considered sufficient to justify their condemnation.

The British Pharmacopœia (1932) gives the limit of lead in the above chemicals as 20 parts per million. There have been, however, vast improvements in the production of the above articles, and the acids can now be obtained with lead content of less than 2 parts per million.

With regard to the use of Artificial Colouring Substances. The Public Health (Preservatives, etc., in Food) Regulations of 1925 prohibits the use of metallic colouring matters and compounds of lead for colouring food.

Food colouring materials are used in small amounts in many food products. The colours are carefully prepared for these purposes and standardised as regards tinctorial power; they generally contain traces only of arsenic and deleterious metals such as lead and copper. In the main it may be stated that colours for foodstuffs contain less than 5 parts of arsenic per million and less than 50 parts of lead per million, whilst many fall below these limits.

The Society of Public Analysts reported in 1925 on the lead content in food colouring materials and gave a list of 14 different colours used in food. Eleven of these contained less than 40 parts of lead per million and most of them less than 20 parts.

4. The spraying of fruits and vegetables with insecticides containing lead compounds.

From time to time lead arsenate and other lead compounds have been found in excessive amounts in the wrappings and skins of imported apples and pears. In one case, two-thirds of a grain

per lb. was present in the wrappings and one-twelfth of a grain per lb. in the skins. These chemicals are widely used as insecticidal sprays on fruits and vegetables and mixed with substances to prevent them being washed off by rain. It was found at first, however, that brushing and wiping would not remove the poisonous residues, but later improved methods were adopted for cleaning the fruit before shipment, which have been effective in reducing the lead content to minute proportions.

The following is an extract from a circular issued in 1935 by the United States Department of Agricultural Food and Drug Administration, Washington, D.C. : " Lead Arsenate Sprays—While lead arsenate sprays are no longer necessary for controlling the insect pests of vegetables, there is as yet no less toxic substitute in the production of apples and pears. However, the commercial cleaning of fruit has become practically universal, and has been perfected to the point where the intake of poison from this source is very much less than at any time in recent years. With continued Federal and increasing local vigilance, the danger to health will never again become significant. We have advised consumers that if they wish to make assurance doubly sure, they may remove any last vestiges of poison spray that may be present by cutting out the natural ' cups ' of the fruit at stem and blossom ends and discarding the peel."

5. Lead in shell-fish and crustacea.

Chapman (1926) made investigations into the presence of lead in shell-fish and crustaceans. He was of opinion that the lead was derived from the sea-water.

Shell-fish and crustaceans examined in the laboratory of the Ministry of Health yielded the following results :

| | Lead in Parts per Million. | | Lead in Parts per Million. |
|---|---|---|---|
| Oysters | 0·2 | Crab | 0·3 |
| Lobster, shell | 3·4 | Winkles | 1·5 |
| Whelks, A | 0·7 | Shrimps (in aluminium container) | 0·3 |
| Whelks, B | 2·1 | | |

## Legislation

The Food and Drugs (Adulteration) Act of 1938 gives added powers for regulating the composition of articles of food or of substances intended for use in the composition or preparation of food.

The Factories Act (1937), Part III—Welfare, enforces the pro-

vision of adequate and conveniently accessible washing facilities for persons employed in factories.

Persons must not be permitted to partake of food or drink where lead is used so as to give rise to any dust or fume.

## Aluminium

Probably no metal has caused so much controversy as the question of the toxicity of aluminium and its salts. While the extensive investigations and experiments carried out from time to time by numerous observers to ascertain the amount of contamination of foods cooked in aluminium vessels and their effect on the human system have frequently given negative results, statements continue to appear questioning the wholesomeness of repeated ingestion of this metal and its salts. The issue is raised again and again by those opposed to its use, and consequently the literature on the subject has become voluminous.

Plagge and Lebbin (1893) conducted experiments in their laboratory. For eighteen months the midday meal, which consisted of coffee and vegetables, was prepared and cooked for two men in aluminium vessels. No metallic taste was noticed, the vessels proving satisfactory. The two men put on weight and remained in good health. The observers concluded that aluminium plates are attacked by most foods, but the amount of the metal taken by a person in a day is only a few milligrams.

In 1913 *The Lancet* published the results of investigations upon the effects of cooking foods in aluminium vessels. The experiments were carried out under conditions similar to those found in ordinary kitchens. Various foods and beverages were cooked in aluminium vessels and the amount of the metal found in the food estimated and the effect upon the utensils studied. The report concluded : " We are confident that aluminium, as it is now made by reputable manufacturers, is a suitable material for cooking vessels, and that any suspicion that it may communicate poisonous qualities to food in the process of cooking may safely be dismissed in view of the results of the practical experiments which we have recorded, showing that the metal is not appreciably acted upon in cooking operations. This finding is satisfactory also, inasmuch as aluminium is an excellent heat conductor ; cooking in aluminium vessels is, therefore, rapid, and fuel is economised in consequence. But the management of aluminium cooking utensils requires the same ordinary applications of common sense as are customary in case of other metals employed for a similar purpose."

Thieme (1929) demonstrated by tests on experimental animals the suitability of pure aluminium vessels for culinary purposes. His experiments extended over several months, and he concluded that abnormally large doses of aluminium salts are devoid of deleterious physiological action.

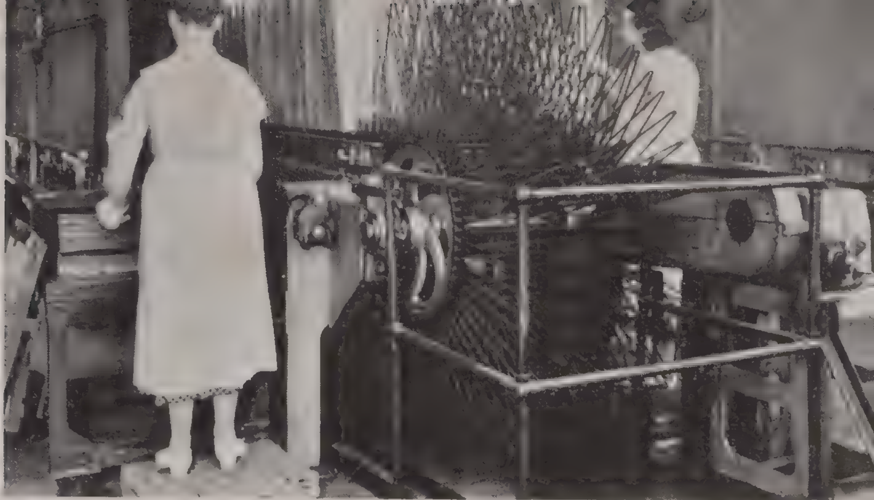
Monier-Williams (1935) in a special report to the Ministry of Health on " Aluminium in Food " arrived at the following conclusions : " Much of the experimental work which has been carried out to ascertain whether aluminium in food is harmful or not is conflicting and inconclusive. Aluminium salts, in doses which are not unreasonably high, have been shown to be not without action on digestive processes. It is a safe rule to exclude from food as far as possible anything which may reasonably come under suspicion of causing harm, and on this account it is undesirable to admit aluminium in the relatively large amounts in which it may be employed as a constituent of baking powders or self-raising flour.

" There is, however, no convincing evidence that aluminium in the amounts in which it is likely to be consumed as a result of using aluminium utensils has a harmful effect upon the ordinary consumer. It is possible that there may be individuals who are susceptible to even such small doses of aluminium as may be derived from aluminium utensils, but evidence of this is inconclusive."

With reference to the addition of alum to flour for the purpose of arresting fermentation or renovating flour damaged by damp storage, this was forbidden by the Bread Acts of 1882 and 1886.

Under the Sale of Food and Drugs Act of 1875, several prosecutions took place as a result of using sodium aluminium sulphate—commercial baking powder—on the ground that it caused gastric troubles. As a result phosphate powders took the place of alum baking powders, but attempts have been made to revive the trade of alum baking powders. Investigations on experimental animals, however, have shown that such baking powder interferes with growth and with the reproductive functions.

During recent years the use of aluminium vessels in food factories has increased. Metal cans are in prominence, especially for fish products which are neither too acid nor too salted. The cans are lighter and do not blacken when used for canning crustaceans and molluscs, etc. No lacquer is required. Fish packed in oil in aluminium containers, however, sometimes develop hydrogen, resulting in a ' blown ' condition.



*Courtesy of the Metal Box Co., Ltd.*

FIG. 17. Lacquering Tin-plates Emerging from the Oven.

FIG. 18.

FIG. 19.—Untrea and treated cor beef cans, ope three months a packing.
The untreated shows considera staining, but treated can is bright as when ginally packed.

[*Courtesy of The International Tin Research and Development Council.*

FIG. 20 (below).—These cans c tained fresh peas. The stained was of plain tin-plate; the lo can had a protective film.

## Tin

The wide use of tin-plate for the construction of receptacles for the preparation, storage and canning of foodstuffs and tinfoil for the wrapping of perishable articles, makes contamination from these sources of considerable interest to food manufacturers. Chemical changes take place between fruit juices when heated in the presence of tin, and the metal is especially taken up by foods containing acids, such as meat extracts, vegetables, vegetable soups, and fruits including peaches, apricots, pears, cherries, pineapples, asparagus and tomatoes. For some reason sardines, silds, herrings and similar fish canned in oil or tomato sauce are particularly prone to attack the surface of tin-plate, and 2 to 8 grains to the lb. have been found present. In some cases the containers were almost completely de-tinned, the fish actually sticking to the inside of the can. The formation of sulphides of iron and tin from the sulphur in certain foods sometimes forms a bluish sheen or marbled appearance on the tin. No corrosion is indicated, in fact a film of such sulphide seems to provide protection during storage against acid juices.

In this connection it may be of interest to mention a process recently introduced for preventing the sulphur staining of the inside of cans and their contents by producing an invisible protective oxide film on tinned plate (as an alternative to lacquering) by immersion in a hot chromic acid solution after preliminary degreasing, or by immersion in hot alkaline phosphate-chromate solution which simultaneously degreases and films the tin-plate (Kerr, 1940). The film, besides preventing discoloration of the tin-plate by sulphur-containing foodstuffs, prevents, to some extent, bleaching of the artificial colouring which is added to certain canned foods. The films produced in the alkaline solutions inhibit rusting of the tin-plate at discontinuities in the tin coating. Cans are less liable to rust during storage or transport, even where climate conditions are humid.

The process was evolved in the laboratories of the International Tin Research and Development Council and trials were made by the Fruit and Vegetable Preservation Research Station (University of Bristol), Campden, the British Food Manufacturers' Research Association, and several canners. These trials have shown that the treatment is most efficacious with meat cans, kidney soup, brawn and meat galantine cans. A fair measure of success also has been achieved with vegetable packs. In this country, development has been held up by the war, but in

view of the keen interest expressed by meat packers, particularly in South America, New Zealand and Australia, designs are being worked out for automatic plant for the filming treatment of cans and tin-plate sheets.

A considerable difference of opinion seems to exist regarding the toxicity of tin. Apparently there is no reliable data that the metal is harmful. Most investigations point to the view that tin does not ordinarily affect the human system.

Regarding the results of various animal experiments carried out from time to time by observers, Schryver (1908) concluded from all the different investigations that " they do not indicate much probability of serious risk of chronic poisoning by the absorption of non-irritant compounds of tin as a result of diet which consists largely of canned foods and is continued over considerable periods of time."

It is generally agreed that foods consumed within a few months of canning may contain as much as ½ grain of tin to the lb., but this does not ordinarily cause gastro-intestinal irritation in the amount usually taken at a single meal. Savage (1920) says : " Tin may exert a toxic action in two definite ways. The amount taken into the body with the food may be so considerable that a single dose may set up acute symptoms or chronic poisoning may be induced by much smaller quantities taken over a long period."

In the view of Buchanan and Schryver (1908), " it seems clear that, in any kind of canned food, quantities of tin approximating to 2 grains to the lb., are not only unusual and unnecessary, but must also be regarded with grave suspicion in consequence of the risk of irritant action of the tin they contain."

Buchanan also drew attention to the desirability from an administrative point of view of requiring the date and place of preparation to be shown on the labels or to be otherwise available when required.

Considerable apprehension exists in the minds of many concerning the presence of metals in food wrapped in foil, although this protects the food from bacteria and dirt. In 1929 the Ministry of Health drew attention to the increasing practice of wrapping foods, such as soft cheeses, confectionery, etc., in tinfoil and to the possibility that, in some cases, excessive amounts of tin may be taken up by food. It was suggested that manufacturers should give their attention to the matter with a view to substituting grease-proof paper or similar material for tinfoil.

The Annual Report of the Chief Medical Officer, Ministry of

Health, 1932, states : " In Hammersmith a number of soft cheeses wrapped in tinfoil were found to be badly blackened and mouldy and were seized and destroyed. A further sample was taken under the Sale of Food and Drugs Act and was found to contain 14 grains of tin per lb. A conviction was obtained."

No standards are laid down for metallic impurities in wrappers ; the matter is a difficult one to decide. It has been suggested that probably the best method of fixing limits is to consider the area of the paper used, rather than the weight, correlating this as far as possible with the amount of foodstuff and the exposed surface of this also.

## Zinc

There is little evidence that the continued ingestion of small amounts of this metal has any deleterious effect on man, and despite the fact that outbreaks of food poisoning are now and then attributed to the contamination of foodstuffs by the metal, experiments have usually not confirmed these toxic properties.

Clayton (1933) remarks : " The normal intake of zinc per day in food by an adult is about 15 mgm., and the normal adult excretes zinc in the urine (0·2–52·0 mgm.) and fæces (2·67–19·9 mgm.). Human milk has been found to contain 3·89 p.p.m., and cow milk 4·58 p.p.m. of zinc. Analyses of Bertrand and Benzon (1928) showed zinc content in p.p.m.—potatoes 5 : garlic 10 : onion 13·8 : peas 44·5 : cereals 12–19·5 : lentils 24·4 : polished rice 2·5."

The use of galvanised vessels in modern food factories is practically unknown, but vessels lined with zinc are sometimes used for the storage of foods. Acid foods are able to dissolve considerable amounts of the metal from galvanised vessels. Investigations made by Sale and Badger (1924) on the effects of various liquids in zinc vessels is shown in the following table :

One gallon of liquid (except in the case of milk, 1 quart) was placed in a galvanised iron pail.

| | *Zinc as Parts per Million.* | |
|---|---|---|
| | After 17 hours' Contact. | 41 hours' Contact. |
| Tap water | 5 | 21 |
| Distilled water | 9 | 27 |
| Carbonated water | 193 | 181 |
| Milk | 438 | 1054 |
| Orangeade | 530 | 854 |
| Lemonade | 1411 | 2700 |

Poisoning by zinc and its salts is not unknown and occasional outbreaks have been recorded, one of which occurred in Surrey in 1922. Two hundred persons were served with apples which had been stewed in galvanised iron pans and all suffered from dizziness, vomiting, colic and diarrhœa. The illness only lasted a few hours and all recovered. It was estimated that each person consumed zinc equal to about 20 grains of sulphate of zinc.

Hereford (1943) reported cases of zinc poisoning caused by the consumption of apple rings which had been cooked in galvanised iron steamers.

" Within three-quarters of an hour of breakfast at an A.T.S. depôt the majority of the auxiliaries who had taken the meal in two adjacent canteens became violently sick. The vomiting was followed in many cases by diarrhœa, cramps, and a varying degree of collapse. All recovered within 24 hours.

" Prompt action by the Medical Officer secured small portions of (*a*) steamed or boiled fish, and (*b*) steamed apple rings, which were sent to the laboratory. A bacteriological examination having failed to yield any suspicious finding, the possibility of metallic poisoning was explored. It was then found that a watery extract of the apple gave a strongly positive reaction for zinc, using a ' spot test ' with acridine hydrochloride.

" Professor Delafield kindly examined the remainder of the apple, which had now been triturated with equal parts W/V of water, and reported zinc present in a concentration of 0·12 per cent., which expressed as crystalline sulphate equals 0·53 per cent. It was therefore evident that a helping of the apple rings weighing 4 oz. would contain about 1·0 g. of zinc in terms of the sulphate, the emetic dose of which is said to be 0·6 to 2·0 g.

" Inquiry revealed that auxiliaries attached to two cook houses had been affected, and that the preparation of the apple had not been identical in both cook houses. In one, after soaking overnight in large mess-tins, the apple rings had been transferred to a skep of a galvanised iron steamer for cooking ; in the other, the tins were placed in the steamer direct. It appeared probable that, in the former method of treatment, zinc would be readily taken up during the cooking, but it was difficult to account for the access of the metal in the latter."

With regard to foods wrapped in zincfoil, Fairall and Walker (U.S.A. 1929), after examining a large number of these, stated that the amount of the metal taken up was very slight, and the normal

degree of contamination constituted a negligible factor in the daily intake of zinc.

The following is the interesting summary by Tanner (U.S.A. 1933), after reviewing evidence on the toxicity of metals and their salts in food: "One is forced to the conclusion that, with the exception of lead and arsenic, the case is not convincing. The evidence in the case of lead leaves much to be desired. There has been too much of arguing on the basis of results secured by injecting pure solutions or feeding them to experimental animals without mixing in food. Furthermore, the metals have been administered in such large doses, in some experiments, that the results are of little value in food poisoning. The point is overlooked that some of the metals may have a beneficial influence on man. Until more is known there is little reason for permitting undue metallic contamination. The metals should be kept as low as possible. The form in which the metal exists is also important. When they exist in foods, they are apparently bound as proteinates and are probably not available for poisoning tissue until they have been liberated."

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## CHAPTER IX

# POISONOUS PLANTS

It has long been known that certain plants, when consumed, are poisonous to human beings. In recent times, however, poisoning of this kind usually has been accidental, the particular plant being gathered and eaten in ignorance of its nature, or in a mistake for some harmless variety. The majority of cases occur amongst children who are especially liable to eat the attractive leaves or berries, including seeds of the peach and bitter almonds. The number of such plants in this country is comparatively few. Some are rare, others are only noxious at certain periods of the year. In a few instances parts of the plants only are poisonous.

The most common are the following: Hemlock (Conium maculatum L.); Cowbane or Water Hemlock (Cicuta virosa L.); Water Dropwort (Oenanthe crocata L.); Monkshood or Aconite (Aconitum napellus L.); Deadly Nightshade (Atropa belladonna L.); Foxglove (Digitalis purpurea L.); Henbane (Hyoscyamus niger L.); Black Hellebore (Helleborus niger L.); Bittersweet or Woody Nightshade (Solanum dulcamara L.); Fools' Parsley (Aethusa cynapium L.); Bryony (Bryonia dioica L.); Laburnum (Cytisus laburnum L.); Black Nightshade (Solanum nigram L.); Spurge Laurel (Daphne laureola L.); Annual Mercury (Mercurialis annua L.); Dog's Mercury (Mercurialis perennis L.).

The above plants contain poisonous substances or alkaloids, the chief being strychnine, atropine, coniine, aconitine, hyoscyamine, scopolamine, solanine and cytisine.

*Hemlock* (Conium maculatum L.). This noxious biennial plant which grows on waste ground, in banks and hedgerows and by roadsides and streams, is widely distributed and flourishes especially in the North of England and Yorkshire. The tall glossy stem is hollow and marked with purplish-red spots, and the leaves are large, somewhat resembling parsley. The small white flowers appear in June and July and are arranged in umbels (as the rays of an umbrella). When not flowering hemlock may be recognised by the appearance of the fruit, each carpel of which has five prominent ridges waved on the margin. The poison (chiefly the alkaloid coniine) is at first present in the leaves, but later is found in the fruits or seeds. The pale yellow root is tapering and not unlike

the parsnip. It is less poisonous than the rest of the plant but varies with the time of the year. It is, however, especially dangerous in its first season's growth. Many cases of poisoning have been recorded through mistaking hemlock leaves for parsley when eaten in salad and in soup ; also from the consumption of the seed and root. Every part of the plant has a strong, disagreeable mousey odour.

The noxious property of hemlock was well known to the ancients, and history relates that the juice from the plant was administered to the Greek philosopher, Socrates.

According to Henslow (1901), " That the poisonous property is not destroyed by boiling is confirmed by a case of two soldiers who collected herbs for boiling with bacon. They partook of the broth, and then of the herbs and bacon. They died in about three hours." In another instance children were poisoned by blowing whistles made from twigs of spotted hemlock.

*Cowbane or Water Hemlock* (Cicuta virosa L.) flourishes in damp situations, such as the edges of ponds, ditches and rivers and is common in temperate climates. It is poisonous to man both in the fresh and dried state. The stem of the plant is stout, hollow, furrowed and branched, not unlike celery, with large dark green leaves, the segments of which are long and narrow.

The seeds resemble anise. Small white flowers appear from July to August. The short tapering thick root which contains a yellow juice is fleshy and hollow, and is often mistaken and eaten for wild parsnip, horse-radish or Jerusalem artichoke, sometimes with fatal results. The plant has a disagreeable mouse-like odour, and all parts are poisonous, especially the root-stock. A small portion of the root or leaves causes a burning pain in the stomach, vomiting, giddiness, convulsions and sometimes death.

Many instances incriminating cowbane are on record. In 1901 a party of boys were camping out on an island in the Firth of Clyde and 24 of them were poisoned by eating this plant.

Gompertz (U.S.A. 1926) reported an outbreak in a Connecticut institution of 17 simultaneous cases in boys who had eaten roots, leaves or flowers growing near their playground. Less than two hours later all were violently ill, with vomiting and convulsions. They received medical aid at once and entirely recovered the next day, with no remembrance of the illness.

*Water Dropwort* (Oenanthe crocata L.), another member of the hemlock family, grows in ditches, marshes, on the banks of rivers and in other damp situations. All parts of this perennial plant

FIG. 21.—Hemlock.

FIG. 22.—Fool's Parsley.

FIG. 23.—Cowbane or Water Hemlock.

Fig. 24.—Deadly Nightshade.

Fig. 25.—Foxglove.

are poisonous, but especially the fleshy, juicy root, which is a spindle-shaped tuber and the chief seat of the poison. It is sometimes mistaken for the parsnip. The tall stem is grooved, branched and hollow with large compound leaves, having divided leaflets ; the latter sometimes cause it to be mistaken for wild celery—when the plant is not flowering. The clusters (umbels) of small yellowish-white flowers appear during July. The fruit is narrow and oblong.

Holmes (1902) gave it as his opinion that water dropwort is the most dangerous and virulently poisonous of all our native species. Its effects are rapid and fatal within a few hours after the ingestion of a small piece of the root.

Sowerby and Johnson (1861) record the poisoning near Woolwich of 17 convicts, who gathered and ate the weed, mistaking it for celery and parsnips. Nine suffered from convulsions and 6 died. The symptoms were tetanus, delirium and insanity.

*Monkshood* (Aconitum napellus L.), Aconite, Wolf's Bane or Blue Rocket, is a perennial plant, the poisonous nature of which was well known to the ancients. It is common in river valleys in South Wales and Yorkshire. It is not, however, usually found outside cultivated gardens.

The plant is about 3 or 4 feet high and grows in circular patches. In the spring it is recognised by the glossy bright green, deeply fingered leaves, which appear before the tall leafy stems. The dark blue or purple flowers, variegated with white, appear in from July to September. The upper sepal of the flower resembles a helmet or monk's cowl. The root is conical or spindle-shaped, pale brown in colour on the outside and white inside and of a fleshy nature, which distinguishes it from the cylindrical pungent root of the horse-radish, with which it is often confounded, resulting in cases of poisoning. The leaves also have been eaten as a salad with fatal results.

All parts of the plant are noxious and the action of the poison is rapid. The taste is bitter and is followed by a burning sensation and numbness accompanied by great salivation, tremors and paralysis.

Henslow (1901) remarks : " So acrid is the poison that the juice applied to a wounded finger affected the whole system ; not only causing pains in the limbs, but a sense of suffocation and syncope." The virulent properties of Aconite (Aconitine), however, depend to some extent on the age of the plant and the climate in which it is grown.

Sowerby and Johnson (1861) say: "Its frequency in the garden and the careless manner in which its deadly roots are often distributed have induced us to place it at the head of our list of British poisonous plants. The recent accident in Scotland, where 3 persons died in consequence of the roots of the monkshood being brought in by a boy from the garden as horse-radish and used by the cook, unconsciously, in preparing sauce for beef, added to many orders of a similar kind, ought to render gardeners cautious in planting and teach them to avoid placing this and other poisonous herbs in the vicinity of those employed for culinary purposes."

*Deadly Nightshade* (Atropa belladonna L.), which is sometimes called Dwale, Barnewort, or Naughty Man's Cherry, is a well-known and a widely distributed perennial plant and grows in chalky soils, on waste ground, borders of fields and in hedgebanks, especially on the North and South Downs and the Cotswolds. It is extremely poisonous (acrid narcotic) to man, but like many other noxious plants there are seasonal variations. The cultivated is less poisonous than the wild variety.

Its noxious properties and fatal effects seem to have been long known, and it is supposed that Dwale was the poisonous plant which occasioned such disastrous consequences to Roman troops under Mark Antony at their retreat from the Parthians.

The downy leaves of deadly nightshade are large, oval and pointed, and arranged in pairs, one of each pair being smaller than the other. The bell-shaped flowers are of a dull brownish-purple colour and appear from June to August. A little before flowering the stout whitish-coloured fleshy root, which is the most noxious part of the plant, is richer in the poisonous principles (hyoscyamine and scopolamine) than after flowering. In August and September the shiny ripe juicy purplish-black berries containing seeds are tempting in appearance and have a somewhat sweetish taste. They resemble small cherries and are especially attractive to children, who are more susceptible than adults to the poison. The consumption of three or four berries causes great excitement, 'double vision,' delirium and stupefaction, sometimes terminating in death. Even half a berry has proved fatal.

It is recorded (Henslow, 1901) that a remarkable outbreak of poisoning occurred in 1846, due to the berries of deadly nightshade being sold in the streets of London as an edible fruit by some ignorant dealers. Two persons died.

A curious case of poisoning by 'belladonna leaves' is described

by Hope (1921)—quoted by Savage and Bruce White (1925): "On 8th May, after partaking of roast stuffed breast of mutton and potatoes, a lady and her two daughters became ill. Their symptoms were dryness of the mouth, giddiness, weakness of limbs, and disturbance of vision and started about ten minutes after eating the food. Belladonna poisoning was diagnosed. The mutton was stuffed with breadcrumbs, salt, pepper, mint, sage and onions. The chemical examination showed that the portions of meat and sage stuffing examined weighed 3 oz. and contained $\frac{1}{40}$ grain of atropine. The dried herbs were obtained from the district of Evesham, and inquiry of the Worcestershire County Medical Officer elicited that the belladonna plant was at one time largely grown in the vicinity of Evesham, but the industry had entirely ceased since 1918. Although its cultivation had ceased and the roots had as far as possible been destroyed, odd plants still continued to come up as the root is very difficult to eradicate. Evidently some belladonna leaves had become mixed with the herbs sent."

*Foxglove* (Digitalis purpurea L.), Throat Wort or Deadman's Bells, is a handsome flowering biennial plant which is found in cleared woodlands and hedgerows on siliceous soils. It is very widely distributed and has long been known as one of the most powerful of our wild poisonous herbs. The cultivated plant is less poisonous than the wild variety. It has an erect stem 3 to 4 feet high covered with grey down. The large downy leaves are of a dull green colour and terminate in a long one-sided bunch of spotted crimson or purple pendulous or bell-shaped flowers which bloom from July to September. All parts of the plant are poisonous (active principle digitalin), especially the seeds, fresh or dried. The leaves are more noxious before flowering than afterwards. In large doses the poison causes vomiting, purging and fainting, and may prove fatal. Foxglove is the source of the well-known drug digitalis which is widely used for medicinal purposes.

*Rhubarb* has frequently been the cause of poisoning, both at home and abroad. The stalks and leaves contain 0·2 to 0·4 per cent. of oxalic acid.

Rosenau and his associates (Harvard Medical School) record a small outbreak due to rhubarb leaves to illustrate their fallacies in diagnosis of 'ptomaine poisoning.' "A female, aged 58, ate about a half-peck of cooked rhubarb leaves (tops) on 14th May, 1917, at 4 p.m. Also took much of the water in which the leaves were cooked. She was sick all night; started to vomit about

3 a.m. on the morning of the 15th. At the same time diarrhœa commenced and continued during the night, but bowels did not move again until the time of death. Patient vomited throughout the entire illness, was very thirsty, and drank a great deal of water. Temperature normal. Patient had had chronic pains in abdomen for many years, which were much intensified during this period. Died in ten days. Rigor mortis did not set in until 30 hours after death. Bacteriological examination of material obtained post-mortem by rectal swab showed that the case was complicated with dysentery.

"A brother-in-law of the patient ate some of the greens and was sick all night, but recovered. A sister ate a very small amount of the greens, and had but slight malaise. Diagnosis: Oxalic acid poisoning."

During the food scarcity in the Great War the use of the leaves was recommended as a substitute for green vegetables (*Lancet*, 1917). The recommendation was, however, soon withdrawn as a number of deaths resulted from poisoning. In 1917 a warning was issued against using soda when cooking rhubarb. Most of the cases of poisoning have been the result of the use of the leaf stalks. Burton (1910), who observed two cases of poisoning caused by eating stewed rhubarb, stated that some people may be more susceptible and others more resistant to oxalic acid poisoning. The patients suffered from diarrhœa, prostration and purging.

Benson (1919) records an outbreak of canned rhubarb poisoning. Nine cases occurred in one family. All were violently ill and two had convulsions. The symptoms appeared about two hours after the consumption of the rhubarb, all the patients recovered.

Tanner (1933) remarks: "In certain parts of France rhubarb leaves are eaten in place of spinach. This custom has caused some serious cases of poisoning. The symptoms appeared in a few hours after the meal, and included pains in the stomach, diarrhœa and cloudy urine of a mahogany colour with large amounts of albumin and cells."

Poisoning by the ingestion of bread made from wheat contaminated by the seeds of certain weeds occurred in South Africa; it was known as "bread" poisoning. The suggestion was that the seeds found their way into the wheat when the threshing machines and mills were not fitted with efficient winnowing and sieving apparatus. In most cases the weed was identified as Senecio, commonly known as ragwort, of which there are several species.

Ragwort is a scheduled noxious weed in this country, and its destruction may be made compulsory under an order of the County Agricultural Committee.

## Edible and Poisonous Fungi

From the earliest times we find records that man regarded fungi as a possible source of food. History relates that the eating of them was a popular custom amongst the ancient civilised peoples. The Greeks and Romans at their feasts and banquets indulged in the consumption of many different varieties, the Boleti being in special favour, Truffles coming next in esteem.

One of the earliest attempts to classify fungi was made by the Greek physician Dioscorides Pedanius, who divided them into the edible and the poisonous. There are numerous references in classical writings to the ways of distinguishing these two groups.

Ford (U.S.A. 1909) who compiled an interesting historical review of the subject, carried out in conjunction with his coworkers valuable work on this type of food poisoning.

Edible fungi now constitute an important article of diet both in this country and abroad. The field mushroom (Psalliota campestris), horse mushroom (Psalliota arvensis), and especially the cultivated variety, are at times in considerable demand, both for edible and canning purposes. Of late years the cultivation of mushrooms commercially has become a recognised industry. Their food value, however, is small.

Merrill (1916, quoted by Jordan, 1931) recorded an instance where a poisonous species grew in a mushroom bed almost to the exclusion of the common cultivated variety and was eaten by five members of the grower's family with almost fatal results. Poisonous mushrooms may apparently develop from commercial spawn, and growers must be careful to eat or sell from the beds only the common mushroom with white cap and pink gills.

Regarding fungi poisoning in the United States of America Jordan (1931) remarks: "There is reason to believe that mushroom (or 'toadstool') intoxication in the United States has occurred with greater frequency of late years, partly on account of the generally increasing use of mushrooms as food and the consequently greater liability to mistake, and partly on account of the increase in immigration from mushroom-eating communities of Southern Europe."

The cultivation of the mushroom began in France and was described by Tournefort in 1707; later, very large quantities

of edible fungi of several varieties were grown near Paris in underground caves some miles in length.

Paulet, who made a study of the incidence of fungi poisoning, records that in the environs of Paris between 1749 and 1788 there were at least 100 deaths. Guillaud (1885) believed that about 100 deaths were thus caused annually in the South of France. Ford (1923) mentions that approximately 1000 cases with 318 deaths and 171 cases with 49 deaths had been recorded in the medical literature of France, Germany and Austria respectively. The same writer reported the occurrence of at least 217 authentic cases with 91 deaths in the United States of America during the past 30 years. Out of more than a thousand species of mushrooms described in the United States over 80 were proved definitely poisonous (Jordan, 1931).

The study of edible and poisonous fungi has been pursued by many workers, and French scientists have done much to increase our knowledge of the subject.

It is recorded that the first systematic investigation was carried out in 1791 by Bulliard, a French mycologist ; he gave the name of " Destroying Angel " to the species Amanita verna.

In this country many books and monographs have been written from time to time on the subject. In 1832, James Sowerby, Junr., compiled an illustrated work on mushrooms and champignons. In 1891 and in 1894 Cook published his books on " Edible and Poisonous Mushrooms " in which he mentions 22 species of the poisonous variety.

In 1910 the Board of Agriculture issued a small illustrated handbook on " Edible and Poisonous Fungi." This excellent work, of which several editions have since been published by the Ministry of Agriculture and Fisheries, contains coloured plates and a detailed description of 24 varieties, 15 edible and 9 poisonous. Contrary to general belief, the number of fungi in this country which have poisonous properties are comparatively few.

During the late summer and early autumn (mushroom season) cases of poisoning frequently occur as a result of persons confusing the edible with the poisonous. The degree and severity of the resulting illness is in proportion to the quantity of ingested poison, as the poisonous properties are chemical in nature and vary in potency in the different species. Idiosyncrasy of the individual plays a part in fungi poisoning. Even the ordinary field mushroom disagrees with some persons and may cause intestinal disturbance, especially if not fresh or badly cooked. Price (1927) attributes

four cases of illness, following a meal at which mushrooms were eaten, to the fact that they were decomposed and that some of them had been frozen.

During the Great War, when there was a shortage of foodstuffs in Germany and Austria, the incidence of this type of poisoning greatly increased. Roch (1916) recorded numerous outbreaks in the districts around Geneva. In 1921, owing to the increasing numbers of cases and deaths in France, a publicity campaign was started. The Pasteur Institute exhibited different species of edible and poisonous fungi and made known the precautionary and other measures to be adopted to combat this type of food poisoning. In some foreign countries laws exist regulating the sale of all fungi, and those retailed for consumption are subjected to an official inspection. In America, the whole subject has been carefully investigated by several workers, including Ford, Abel, Bronsen, Patterson and Charles, McIlvaine and Schlesinger, and as a result, much useful information has been forthcoming. Ford (1923) divided the poisonous fungi into groups :

(A) Gastro-intestinalis : those causing gastro-intestinal disturbances of a more or less violent character, but rarely with fatal results. The species chiefly concerned are : Boletus satanas, Lactarius torminosus, Russula emetica, Entoloma lividum and Lepiota morgani.

(B) Choleriformis : those producing the degenerative changes in the internal organs and tissues, loss of weight together with initial gastro-intestinal symptoms followed by violent pain, delirium and coma, with high mortality. Species concerned : Amanita phalloides, A. verna, Pholiota autumnalis and Hygrophorus conicus.

(C) Nervosus : those in which the poisons act on the nerve centres causing profuse perspiration or salivation, followed by delirium, hallucinations, convulsions and coma. In the early stages there is violent gastro-intestinal disturbances. There are, however, many mild cases of this type. Among the many species concerned are Amanita muscaria, A. pantherina, etc.

(D) Sanguinareus : causing gastro-intestinal symptoms followed by jaundice, anæmia and hæmoglobinuria with low mortality. Species definitely incriminated—Helvella esculenta.

(E) Cerebralis : symptoms of transient excitement and hallucinations caused by only two species Panoeolus papiliomaceous and P. campanulatus.

There is no general infallible method of distinguishing edible

from poisonous fungi. Several varieties or closely allied species in this country are edible and wholesome. The only safe procedure is to learn to identify certain well-recognised species by their botanical features, as the field mushroom, or the horse mushroom, and to avoid those growing under trees or in woods, as it is easy to make mistakes with the numerous varieties, some of which sport bright colours and are very poisonous. Even in the case of those known and correctly identified, caution must be exercised. It is essential that they should be fresh and be free from attacks by insects, or other organisms causing decomposition. Mushrooms are indigestible when eaten raw and unwholesome when decomposed.

The distinguishable features of the common mushroom are as follows : Grows usually in short grass in open pastures, uplands or downs, in summer and autumn. In the young or ' button ' stage it is whitish and nearly round. Later the cap expands and becomes hemispherical and nearly flat. The mature cap is white or brownish-white in colour, skin dry, silky, smooth and peels easily and cleanly. Stem white and solid but slightly pithy, enlarged below and requires a twisting movement to break it off. Membranous ring round the middle or towards the top. No sheath near top or at base. Flesh is white, thick and soft, colour changing to reddish or dirty brown when broken or cut. Gills thin and crowded and not joined to stem. Colour whitish in ' button ' stage, but becoming pink and finally dark purplish-brown to black. Odour earthy but not disagreeable. Taste somewhat earthy but pleasant.

The following description of the horse mushroom (Psalliota arvensis) is given in Bulletin No. 23 (1935) Ministry of Agriculture and Fisheries : " This species is larger than the common mushroom, usually 4 to 6 ins. across, though specimens up to 8 ins. across are not uncommon. The cap is at first almost globose, then hemispherical, and finally becomes almost flat. It is whitish in colour and silky-smooth, and becomes slightly stained with pale brownish-yellow when injured. The stem is white, sometimes stained with brownish-yellow, stout, thickened at the base, with a large spreading *double* ring towards the upper part. The gills are at first white, then finally dark reddish-brown. The flesh is firm, thick, white, and sometimes tinged with yellow.

" The horse mushroom is common in summer and autumn in pastures and beneath scattered trees, where it sometimes occurs in large rings, termed ' fairy rings '. It differs from the common mushroom not only in its larger size, but also in the flesh not

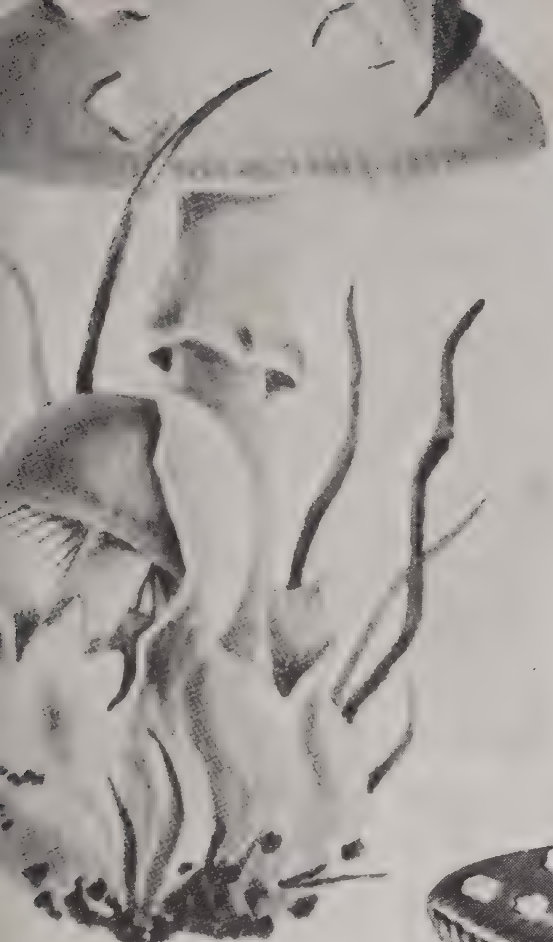


FIG. 29.—The Death Cap.

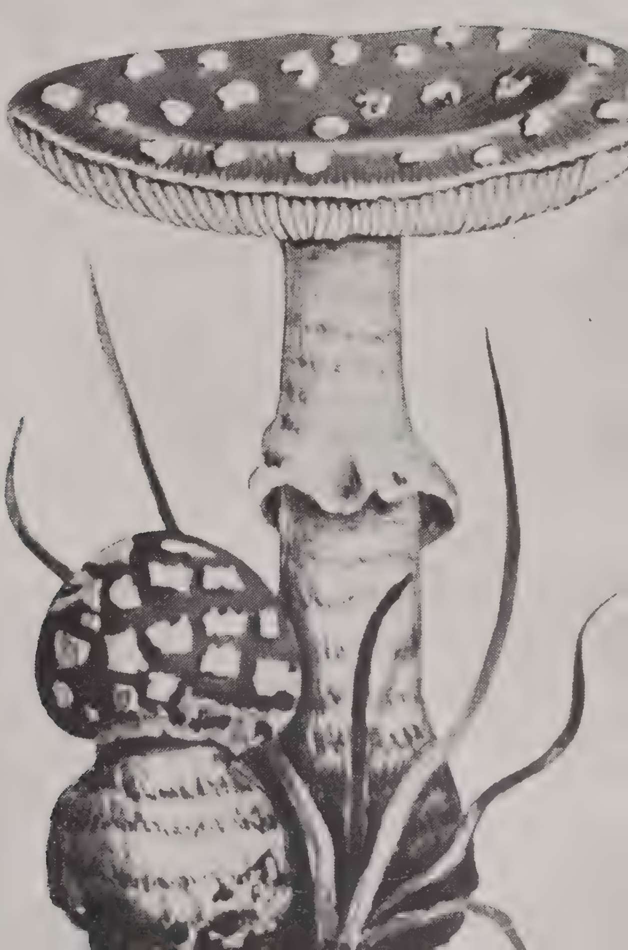
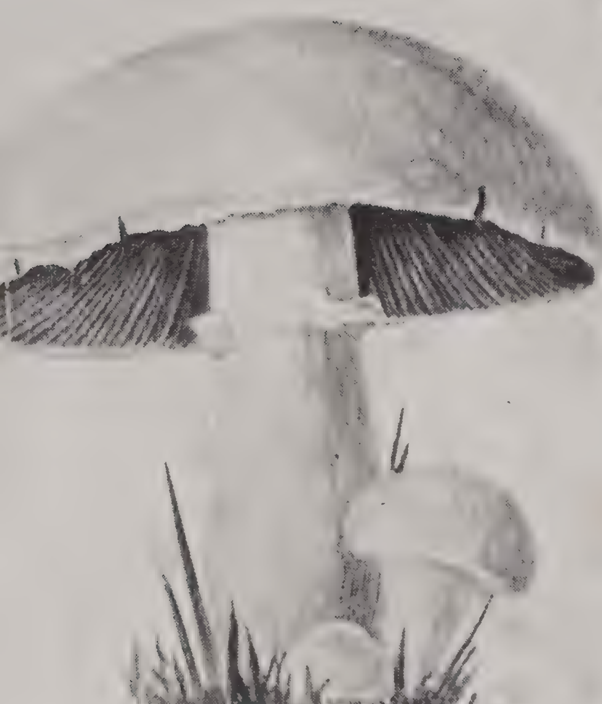


FIG. 30.—Fly Agaric.

Fig. 26.—Mushroom Growing on a Commercial Scale.



. 27. Common Mushroom (Psalliota campestris).

Fig. 28.—Verdigris Agar

becoming brown when cut and in the gills remaining dry when old."

The chief poisonous varieties of fungi found in this country are: Death Cap or Cup, or Deadly Amanita (Amanita phalloides); Bulbous Agaric (Amanita mappa); Fly Agaric or Scarlet Fly Cap (Amanita muscaria); Warted Agaric (Amanita pantherina); Crested Agaric (Lepiota cristata); Glutinous Agaric (Volvaria gloiocephala); Purple Agaric (Cortinarius purpurascens); Yellow-staining Mushroom (Psalliota xanthoderma); Verdigris Agaric (Stropharia æruginosa).

Of the above species the Death Cap and the Fly Agaric are the most frequent cause of poisoning.

The Death Cap is said to be the cause of 90 per cent. of the deaths caused by fungus poisoning. Ford (1909) calculated that 12 to 15 deaths occurred annually in the United States of America from this species alone. Dettrich (1924) estimated that in Germany it caused 80 to 90 deaths every year. It is extremely poisonous, very small quantities of the fungus causing intense suffering and sometimes death. Children are more susceptible than adults. It acts firstly as a digestive irritant. After an incubation period of 6 to 15 hours there is sudden recurrent violent pain in the abdomen, thirst and choleraic diarrhœa. The complexion becomes a peculiar yellow colour (Hippocratic facies), *la face vulteuse* of the French (Ford, 1909). Secondly, it acts as a proto-plasmic poison, causing degeneration of the cells in the liver and nervous system, followed by coma and death. The rate of mortality from 60 to 100 per cent.

The Death Cap is found in woods and adjoining pastures. Greenish or yellowish-olive, occasionally white, in colour, the cap is streaked with dark fibres and is sticky when moist. The stem is whitish and sometimes tinged with green with a loose silky ring towards the upper part. The base is bulbous and is sheathed by a large yellowish-white cap which is more or less buried in the soil. The gills are white with sometimes a slight greenish tinge. The flesh is white with a greenish colour under the outer skin. When old, the fungus has a fœtid odour.

The poisonous properties of A. phalloides were investigated originally by Letellier in 1926. He isolated a substance which he termed 'Amanitin'. Later, several other workers attempted to obtain the active poisonous principle of the fungus, and in 1891 Kobert extracted a powerful hæmolytic poison (acting upon the red corpuscles of the blood, dissolving out the red colouring matter)

which he named 'phallin.' In 1901 the same worker demonstrated a poisonous substance in alcoholic extracts of A. phalloides, and after further experiments concluded that the active principle was an alkaloid.

Ford (1906) investigated these substances and found that phallin (which he termed 'Amanita hæmolysin') lost its hæmolytic property when heated to 70° C. or on exposure to weak acids or alkalis and by the action of pepsin or pancreatic juice. Nevertheless, the substance, after heating, still retained its toxicity for experimental animals and gave rise to lesions similar to those seen in human cases poisoned by the fungus.

Ford and Bronson (1913) considered that Amanita hæmolysin was of little importance in cases of poisoning. They concluded that the extracted heat-resisting substance (named 'Amanita toxin') which was devoid of hæmolytic properties could not be regarded as a protein or glucoside, but was the active principle responsible for the fatal human cases following the consumption of the fungus A. phalloides. It ranked as one of the most powerfully known poisons of plant origin.

Damon (1928) remarks: "From our present knowledge of the subject the active principle in poisoning from this species of fungus undoubtedly appears to be the amanita toxin, with amanita hæmolysin playing but a minor part, if any at all, in the intoxication."

## Illustrative Outbreaks

Plowright (1905) reported several typical cases of poisoning by A. phalloides. One, a boy of 12, ate a small portion of the raw fungus, at 11.30 a.m. About 1 a.m. the next morning (13 hours later) he commenced vomiting and suffered from thirst and diarrhœa. These subsided and he was able to eat his breakfast but soon afterwards the vomiting and diarrhœa returned. Later, however, his condition greatly improved. These periods of attack and remission were repeated until the fifth day when the boy had slight convulsions and died.

Plowright (1905) also records an interesting outbreak which occurred in a family of 4 persons, two of whom were severely poisoned by the fungus, but recovered.

A man, his wife, son and daughter gathered and consumed 4 to $4\frac{1}{2}$ lbs. of A. phalloides. The mother and son ate the fungus in a raw state and early the following morning were taken ill, as were the father and daughter later. The usual symptoms, i.e. thirst, sweating, vomiting, gastro-intestinal disturbance and intense

abdominal pain were observed. The son developed convulsions, distortion of the face muscles, dilation of the pupils, involuntary oscillations of the eyeball, and he died 54 hours after eating the fungus. The mother developed jaundice on the third day and suffered from cramp-like pains. She aborted a 3 months' old fœtus and on the fourth day was restless, with retracted head, almost unconscious, with complete anuria, respiration became irregular. She succumbed about 100 hours after ingestion of the fungus. The father exhibited similar symptoms, but on the eighth day felt better and eventually recovered. The daughter had diarrhœa with blood and mucous in the stools, great thirst and enlargement of the liver, The diarrhœa gradually subsided and she slowly recovered.

This fungus, which causes severe illness and sometimes death, grows under birches and firs and in woods. The name ' fly agaric ' is derived from the fact that a decoction of the fresh fungus was formerly used as a fly poison. It somewhat resembles the edible amanita, but can hardly be mistaken for the common field mushroom as it is a brilliantly-coloured decorative species and is one of the most beautiful of the agarincini. The expanded flat and sticky cap (4 to 7 ins. across) is of a scarlet or orange-red colour and covered with thick irregular whitish warts. The stem is white or yellowish in colour and 4 to 7 ins. high. The base is bulbous and is encircled by several concentric rings formed by the remains of the ' volva ' (a cup or sheath-like structure at the base of the stem).

Amanita muscaria contains the alkaloidal substance ' muscarine ' which has been isolated by several workers. Patterson and Charles (1915) suggested, however, that there were probably other poisons present besides muscarine, because atropine, which was a perfect antidote for muscarine, did not entirely neutralise the effect of injections or decoctions of this species of fungus.

Savage (1920) remarks : " Although muscarine is a powerful poison the symptoms it produces in the human subject are not identical with those produced by this type of mushroom poisoning. Also an infusion of the fresh fungus is very poisonous to flies while muscarine itself is harmless to those insects. While, therefore, it is reasonable to assume that muscarine plays a large part in the toxicity of this mushroom, it is probably associated with other poisonous bodies which have not yet been isolated and studied."

The characteristic symptoms vary considerably in intensity in individual cases, following the ingestion of this fungus. They usually appear in from 1 to 6 hours but shorter incubation periods

have been recorded. There is salivation, sweating, lacrimation giddiness, vomiting and diarrhœa. Respiration is accelerated bu the pulse is slower and irregular.

In most cases the pupils of the eyes are contracted and do no react to light and accommodation.

In severe poisoning, nervous and mental disturbances occur and there is violent gastro-intestinal reaction and later delirium convulsions and sometimes death from respiratory paralysis.

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CHAPTER X

# POISONOUS FISH

This type of food poisoning is not very common in this country, but there are many kinds of fish, especially those found in tropical waters, which if eaten, even when in an apparently healthy condition, sometimes produce symptoms of poisoning—symptoms which are more likely to occur if certain parts are consumed, such as the liver, roe, head or intestines. Fresh fish is usually sterile as regards the interior, but numerous organisms are present on the surface, even of freshly caught fish.

" Fish are covered with a thin layer of a mucous substance, and this increases when the fish dies. It is composed of nitrogenous substances which facilitate the growth of numerous types of bacteria found in sea water and in fish fæces. Bacteria on and near the gills are an important source of infection that causes spoilage of fish and greatly increase the rate of bacterial decomposition. The intestines of fish do not contain a typical commensal flora, like mammals, but seem to depend upon the type of food ingested " (Shrader, 1939).

Fellers (1926), in a study of raw salmon spoilage, found numerous organisms in the mouth, gills and slime of live salmon. He observed that bacteria penetrated the flesh under average conditions in from 24 to 60 hours, depending upon such factors as the size and species, temperature and methods of handling. The number of bacteria in the flesh increase rapidly with each 24 hours.

The symptoms of poisoning are usually of two kinds : (A) Gastro-intestinal irritation with rapid prostration and sometimes urticaria. (B) Severe nervousness and convulsions.

In some fish a poisonous substance appears to be secreted at certain times of the year. For instance, the roes of pike, sturgeon, carp, bream and turbot produces violent intestinal disturbance if eaten during the breeding season.

Abraham (1906) reported 28 cases of poisoning from the ingestion of infected pike. The symptoms were like those observed in typhoid fever, but examination for ptomaines and poisonous metals was negative. An organism of the aertrycke type, however, was isolated.

Jordan (1931) remarks : " The season of the year at which the fish is taken is undoubtedly a factor of importance, and there is

evidence connecting the presence of toxic constituents with the state of the reproductive organs. The ovaries of the sea urchin, which is eaten by some of the Mediterranean people, are said to be poisonous during the spawning season."

Fish roe poisoning is common in Russia and causes severe gastro-enteritis. Cases of intoxication from infected fish have been recorded by Sieber (1894–5) from Russia.

The initial products of decomposing fish are extremely toxic and attack the nerve centres, producing a type of illness somewhat resembling botulism. The poison is undestroyed by salting, although the putrefying bacteria are killed, and will produce severe poisoning if the salted fish is insufficiently cooked before it is consumed.

Certain varieties of fish are perfectly harmless if eaten as soon as they are caught, but become toxic if allowed to remain uncooked even for an hour. Of the fish ordinarily consumed in this country, mackerel has the worst reputation for occasionally causing illness, possibly due to the rapidity of decomposition ; this fish should be eaten as soon as possible after being caught. Some persons have a peculiar idiosyncrasy to mackerel, and even herrings, and become ill after eating them, although they are in a fresh condition.

According to Günther (1880) the flesh of certain members of the herring family, such as Clupea thryssa and Clupea venenosa, are poisonous. The former—all parts of which are poisonous—has been known to cause death before being actually swallowed.

Anderson (1907) described in detail the tests by which the decomposition of fish can be recognised. The usual signs are as follows : When rigor mortis has passed off. The eyes are sunken and of a grey colour. Gills greyish or muddy white, later becoming greenish and slimy. Flesh along backbone shows a reddish discoloration and is easily stripped from the bone. The degree of discoloration depends upon the time elapsing since the fish was caught. Wall of abdomen soft or pulpy, sometimes showing a jelly-like appearance with discoloration. A tainted or even putrid odour. The scales of stale and decomposed fish have lost their sheen and become detached easily. Rigidity and stiffness are sure guides for fresh fish. Even though they may be well iced, fish soon become stale and begin to deteriorate.

It may be of interest, in passing, to mention that in recent years most of the commercially important species of white fish have been kept experimentally in cold store at temperatures ranging from $-5°$ to $-30°$ C. for periods up to 6 months (Reay, 1929). As a

result of the tests it has been recommended that for periods up to 6 months the freshly caught fish should be rapidly frozen, glazed and stored at temperatures ranging from – 20° to – 30° C. Fish treated in this manner remain in good condition, are highly palatable and, moreover, suitable for smoke curing.

Reay (1935) studied haddock frozen at – 21° C., held for different periods and then thawed and stored in ice, and found that the bacterial flora did not differ from that of fresh fish stored likewise, but that the thawed product deteriorated more quickly.

Among the tropical fish well known to be poisonous are the different varieties of wrasse, the parrot fishes so named from their brilliant colouring, the toad fishes, the file fishes and the family of Tetrodontidæ (globe, puffers and balloon fishes), comprising many important species such as the 'fuga' of Japan, which cause so many deaths among the Japanese. This family is widely distributed along the coasts of Japan, China, East Indies and Africa. Poisoning by these fish is acute and onset of symptoms rapid. The nature of the poison has been studied by Takahashi and Inoko (1890), Micera and Takesaki (1890), Tahara (189 , 1911) and others.

According to Norman (1931), "There are a number of fishes which, although without definite poisonous organs, have their flesh more or less permeated with poisonous substances, taking the form of alkaloids of a particular kind called leucomaines. This may be regarded as a special form of protection, saving the species by poisoning its enemies. To eat certain species such as muki-muki, or death fish of Hawaii, is to invite certain death."

## Shell-fish Poisoning

Many persons show a definite idiosyncrasy to shell-fish generally, even when eaten in season, and urticaria and gastro-intestinal symptoms, etc., which vary considerably in individual cases, usually follow their consumption.

Apart from outbreaks of typhoid fever due to infected shell-fish collected from beds polluted by sewage, cases of food poisoning have been reported from time to time due to the consumption of mussels, oysters, cockles, crabs, lobsters, etc. Mussels are particularly liable to be toxic, and cases and deaths have been recorded (see Cameron, 1890; McWeeny, 1890; Todd, 1891; Hill, 1895; Kofoid, 1927 and Meyer, 1928) as being due to the ingestion of these shell-fish.

Many theories have been put forward to account for their

poisonous nature. Dutertre came to the conclusion that no class of mussel was always poisonous, that the toxic action was not due to some particular food eaten by the mussel, or to spawn or any portion of the mussel itself or to decomposition, but that the poison was due to a true disease attacking the liver.

Dodgson (1928) remarks: "The widely held popular view that all manifestations of poisoning, due to the consumption of mussels, arise from a common cause inherent in mussels is erroneous. Certain popular conceptions that poisonous properties reside in the 'beard,' foot or other particular parts of the mussel, are erroneous."

Meyer (1928), in recording an outbreak of mussel poisoning in California in 1927, where 102 persons were affected and 6 died, points out that poisonous mussels cannot be distinguished from sound molluscs, either by appearance or behaviour on cooking; occasionally a pungent odour may be noticed and the 'liver' is always large and dark. In this particular outbreak the mussels were neither located in stagnant nor polluted basins, but were subjected to the ebb and flow of the tide. Incidentally, poisoning due to the consumption of Pacific Coast mussels has been known since the days of the Indians. They noticed that if the shell-fish were eaten after being collected when the ocean waves were luminescent (in hot weather) they caused illness and death.

Somner and Meyer and their colleagues (1937), who made an experimental study of paralytic shell-fish poisoning comment as follows: "Deductions from analogy with other poisons are of little avail in the elucidation of the problem, since paralytic shell-fish poison seems to belong to a category all its own from the toxicologic as well as possibly from the chemical point of view. In its powerful action of the respiratory centre it resembles some of the most potent alkaloids, but it far surpasses them all in toxicity."

The investigators tabulated 243 cases of paralytic shell-fish poisoning, with 16 deaths, that occurred between Ventura County, California, and Juneau, Alaska, from 1927 to 1936. Of these, 234 were caused by the coast mussel and 9 by the Washington clam.

They conclude: "Poisonous mussels may in no way be distinguished from normal ones except by the animal test. Mussels subjected to various conditions in the laboratory have never shown an increase in toxicity; they usually show detoxification, the rate of which has been determined. Mussels may take up poisons from sea water. Strong evidence has been presented which points to

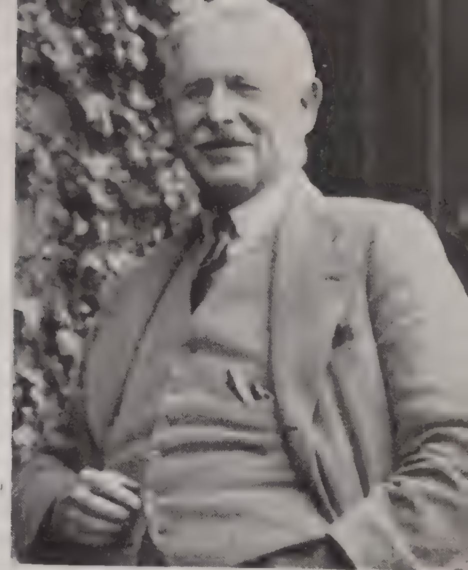

Fig. 31.—R. W. Dodgson, M.D.

Fig. 32.—Oyster Purification Tanks, Brightlingsea, Essex.

Fig. 33. Ministry of Agriculture and Fisheries.
Mussel Purification—Hosing the Mussels.

Fig. 34.—Dr. Ernest C. Dickson, 1881–1939.

Fig. 36.—Professor Karl Meyer, M.D.

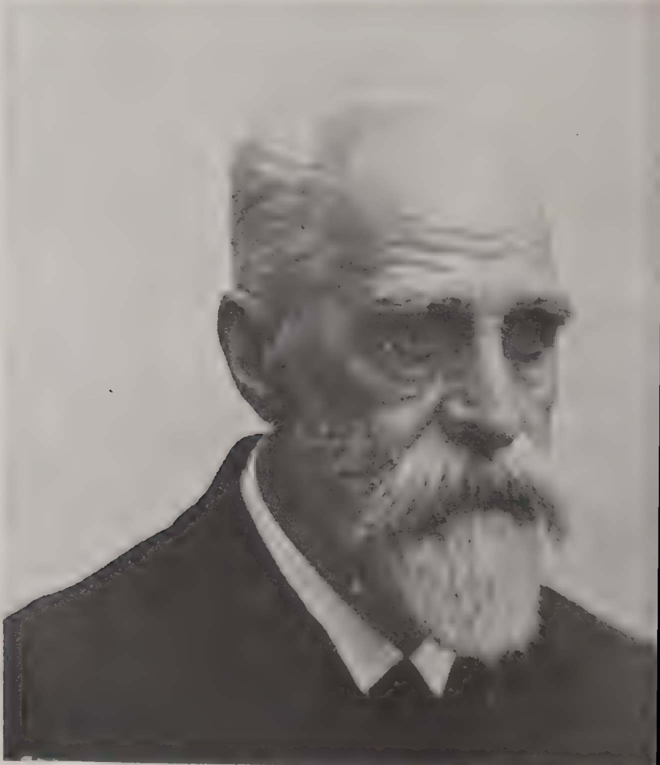

Fig. 35.—Prof. Emilé P. M. Van Ermengem, 1851

the water of the open ocean as a carrier of the poison. Owing to the strong absorption of the substance on base-exchanging silicates of the sand, it is not likely to occur free in the water. The poison has been demonstrated, at least during the poison season, in the residue from filtration of sea-water. Whether it is contained in the plankton or absorbed in the microscopic sand cannot at present be decided."

A summary on "Mussel Poisoning" by Sommer and Meyer (1941) has been published. Regarding the source of the poison they say: "The original source of the poison is found in a unicellular microscopic organism of the ocean, the dinoflagellate Gonyaulax catenella. It is a free-swimming organism, multiplying by formation of chains of 2, 4 or even 8 individuals, of dark orange or greenish-brown colour, and living, like a true plant cell, by photosynthesis. Like all plankton organisms it is most abundant in the summer; at times it may multiply to as large a number as 40 millions per litre. At such times the water may, for miles, present a deep rust-red colour, the so-called 'red-water,' in daytime, and a beautiful luminescen tspectacle at night. Needless to say, other dinoflagellates or diatoms may present similar pure culture developments in the ocean without being poisonous. Gonyaulax catenella may vary considerably in its poison content; even a small number which is not visible as red water may be sufficient to cause dangerous conditions in shell-fish. It occurs in the open Pacific Ocean, less in enclosed bays and estuaries, from Alaska to Southern California. It has been tentatively identified in the North Atlantic Ocean (Nova Scotia and Belgium).

"The strong radiation of the sun together with the cold nutrient waters due to the upwellings along the Pacific Coast in summer time seem to be the ideal conditions for the growth of this dinoflagellate.

"The Poison: The poison contained in this organism is one of the strongest known. It belongs to the class of alkaloids, such as strychnine, muscarine and aconitine. It is heat-stable in acid or neutral solution, but is gradually destroyed by boiling with alkali. It is readily soluble in water and alcohol, insoluble in ether or chloroform. About one millionth of a gram is sufficient to kill a mouse on injection; the fatal dose by mouth for a man is probably a few milligrams. The toxic principle has not been isolated in a crystalline state but has been purified to a high degree in the form of its hydrochloride."

Dodgson (1928) says : " Mussel poisoning includes at least three distinct pathological conditions or types of condition, namely :

" (*a*) ' Musselling ' or the erythematous form, which is due to properties inherent in the mussels. It affects a limited number of specially susceptible people, who should avoid mussels. The symptoms are of short duration, and unpleasant while they last, but are never of serious import.

" (*b*) The paralytic form, always grave and even fatal. Its cause is not definitely known, but it is always due to mussels from foul or stagnant waters. It is extremely rare, some 8–10 cases only being on record. The danger of contracting it may be reduced to such small proportions as to be probably entirely negligible if elementary caution be exercised, and especially if only purified mussels be eaten.

" (*c*) The bacterial food poisoning form. There are recorded in the literature several cases of fatal poisoning following the consumption of mussels, which were, in all probability, instances of bacterial food poisoning. Generally speaking, the cases in question are difficult to classify, either because the information available is inadequate for the purpose or because certain of the symptoms were such that it is not possible to exclude the paralytic form."

With regard to (*b*) Paralytic form, the classical mussel poisoning outbreaks at Wilhelmshaven in 1885 (19 cases with 4 deaths) and Dublin in 1908 (7 cases with 5 deaths) well illustrate this type of poisoning. The typical symptoms were vomiting, swelling of the face, constriction in throat, numbness of mouth and lips, pricking and burning sensation in hands and feet, want of co-ordination of movements, giddiness, spasms and dilation of the pupils of the eyes, death resulting from respiratory paralysis.

In the Wilhelmshaven outbreak, Brieger (1889) isolated a substance he called ' Mytilotoxine ' from the mussels which, when injected into animals, produced all the symptoms of mussel poisoning.

Owing to the successful experiments carried out in connection with the purification of mussels at the Fisheries Experiment Station (Ministry of Agriculture and Fisheries), Conway, North Wales, under the direction of Dr. Dodgson, the process on a commercial basis has now been going on for over 20 years. Oysters are also subjected to similar treatment on a commercial scale at Brightlingsea, Essex.

The system of purification is based on the natural action of the

bi-valves clearing their alimentary canals freely in sterilised sea-water and thus becoming gradually cleansed. They not only get rid of the sewage and the bacteria, but also all particles of solid matter. Bacteriological examination has demonstrated that the bi-valves are freed from possible contamination and fit for human consumption. Thus the problem of safeguarding the public health from infection from these particular shell-fish is practically solved. It should be specially noted that the shell-fish thus purified are not impaired either in keeping quality or any other respect.

Ozonisation treatment for the purification of oysters is also in use at a number of sea fisheries. Briefly, the process consists of passing sand-filtered, ozonised sea-water continuously over trays (fitted in wooden vessels) containing the oysters, which are cleansed in from 48 to 72 hours. The treatment is effectual, cheap and economical.

It may be of interest in passing to mention an outbreak recorded by Gray (1936) at Avonmouth, of acute gastro-enteritis affecting 18 persons. The illness, which was caused by eating cockels purchased from an itinerant vendor, was associated with **B. Proteus vulgaris.**

Lobsters and crabs tend to decompose quickly. The indication of a good and fresh condition in the lobster is a clear, hard shell, with flesh plump and firm. After being cooked, the tail on being pulled out should spring back sharply.

Boiled crabs do not keep well in hot weather. This is shown by the discoloration of the apron from which an indescribable odour issues. The parts beneath the claws become sticky and wet. If the shell appears faded in appearance it indicates staleness.

The shell of a healthy oyster should be tightly closed. If open it should immediately close on being handled, otherwise it is dead and unfit for food. Oysters should be eaten immediately on being opened.

The Acts and Regulations relating to shell-fish are as follows :

Oyster, Crab and Lobster Act, 1877.
Public Health (Shell-fish) Regulations, 1934.
Food and Drugs Act, 1938, Section 39.

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CHAPTER XI

# FOOD ALLERGY [1]

The existence of peculiar abnormal reactions or idiosyncrasies or sensitisation to food has been recognised since ancient times. Many persons are unable to eat certain foodstuffs, even in very small quantities, without exhibiting some characteristic and/or disagreeable symptoms ; moreover, the number of food-sensitive persons is, in all probability, much greater than commonly suspected. The old adage, "One man's meat is another man's poison," probably originated from the knowledge of these idiosyncrasies. Physiologically this peculiar and interesting sensitivity to particular foods, termed 'food allergy,' von Pirquet (1906), which is fairly common and sometimes of a more or less serious nature, is primarily due to the constitutional condition of the individual concerned and not to the result of eating unwholesome foodstuffs.

The foods which commonly produce these allergic reactions usually contain proteins or are protein in nature (nitrogenous foods), and include a large number and variety of articles of diet, such as eggs, milk, cheese, fish, shell-fish, cereals, potatoes, pork, strawberries, blackberries, mushrooms, etc., or a combination of several of these commodities. Moreover, there is some evidence that non-nitrogenous substances (oils, fats and carbohydrates) also may be responsible.

With regard to fish, even the odour of cooking is sometimes sufficient to induce symptoms in highly sensitive persons. Cooking has little effect upon fish allergy. Cereals, which are common ingredients of the average diet, have many commercial uses. They are of special importance because of their ability to cause symptoms, either as a result of inhalation (as asthma in bakers) or when ingested, being capable of producing almost any variety of allergic manifestations.

Rarely is a person sensitive to only a single food, but generally he is so sensitive to a group of similar foods. Investigators who studied the subject extensively, found that next in frequency come

[1] "Allergy is a general term applied to any alteration in the reaction of the living organism to foreign substances ; it may be antigenic or non-antigenic in character" (Jordan, 1931).

chocolate, cabbage, tomatoes, oranges, cauliflower, bananas, walnuts and carrots. The symptoms resulting from allergic reactions, which vary considerably in individual cases, may be mild or severe and include nausea, vomiting, migraine, urticaria, erythema, eczema, or gastro-intestinal disturbance, constipation, and certain types of malnutrition. The onset of the illness may be sudden or delayed some hours. Very mild reactions, which are the commonest, sometimes produce symptoms so slight that their true nature may be overlooked. These mild reactions may also give rise to recurring illnesses, or chronic ill-health.

Foods to which an individual may be sensitive do not always produce the same manifestations. For instance, one may cause an urticarial rash and another a gastro-intestinal disturbance. Occasionally the mere handling of a certain foodstuff, such as flour (or even drugs), by very sensitive persons, is sufficient to set up localised reactions, particularly skin affections, such as eczema. Special exposure enormously increases the incidence of sensitiveness.

Unfortunately several of the above-mentioned symptoms, which vary considerably in individuals, are often present in various types of food poisoning, especially isolated cases with gastro-intestinal symptoms, which are more likely to be due to food allergy, than are large outbreaks, thus adding to the complexity of the whole subject.

Food idiosyncrasy, commonly present in infancy, tends to grow less as age advances, but it may be acquired at any time during life as a result of excessive consumption of some particular or unusual food, such as mushrooms, strawberries, etc. Ratner (1928) suggested that under certain conditions an infant with an allergic predisposition may be sensitised before birth by the mother's over-indulgence in certain protein foods. The condition may or may not be hereditary, and there appears to be a considerable difference of opinion on this subject though hypersensitiveness exhibited towards certain foods is frequently present in parent and offspring. An inherited tendency to become sensitive to certain foodstuffs may show itself at any time after birth, but it does not necessarily follow that descendants will suffer from the same allergic manifestations as their antecedents. The tendency to become sensitive, however, is no doubt transmitted from one generation to another. A case has been recorded (Richet, 1913) where idiosyncrasy to eggs existed in four generations.

In infancy great difficulty is frequently experienced in feeding.

and this is increased by the presence of a sensitisation to common foods, such as milk, eggs and even human milk. Sensitivity to eggs, more than other foods likely to cause allergy, is provocative of infantile cutaneous manifestations such as eczema and urticaria. Children showing an idiosyncrasy to cow's milk can often drink goat's milk with impunity. A definite early history of dislike for, or avoidance of, some particular food or of disturbances caused by them, may sometimes suggest the presence of this hypersensitive condition.

Regarding the general issue of milk to schools, Kennedy (1936) remarks that " the widespread drinking of milk by school children and others may yet have to be considered in the light of food allergy."

A characteristic feature of food idiosyncrasy, especially in young children, is the tendency for one reaction to be replaced by another. It is quite possible for an infant with severe urticarial rash to outgrow this manifestation and in later years to become subject to gastro-intestinal symptoms. In certain individuals the periods of sensitivity may be separated by periods of lessened sensitivity when they are comparatively free from attack. In some persons the abnormal condition may gradually become continuous, whilst in others the reactions may actually disappear altogether in course of time.

Dodgson (1928) described an attack of ' musseling ' he had when a young man. About 24 years later he again ate mussels with the intention of recording the symptoms. Nothing however happened.

Although the real cause of food allergy is probably not fully understood, the abnormal condition has been generally assigned to individual hypersensitiveness to foreign protein substances circulating in the blood. It is well known, as a result of experiments, that the injection of protein substances into man and animals may at times set up poisonous symptoms. Some observers hold the view that this hypersensitive state may be due in the first place to an abnormal permeability of the intestinal mucous membrane, which allows the unaltered proteins to pass through in an unchanged state and in this way gain access to the bloodstream. The condition resembles in certain respects the condition known as anaphylactic intoxication, which is presumed to be an exaggeration of the normal defence of the body against proteins and bacteria through the agency of the processes of digestion.

Savage (1920) remarks : " The hypothesis that these cases of

food idiosyncrasy are a variety of anaphylaxis is based on the supposition that in the individuals who exhibit the condition there is a marked hypersensitiveness to the action of particular proteins in these special foods, that they gain access to the circulation as unaltered protein and that the symptoms caused are due to individual intolerance of their presence in the blood. There are strong arguments which suggest this is the true explanation. In the first place the symptoms induced, including the rapidity of onset (allowing time for absorption from the alimentary canal), the minute dose required and the lesions caused, resemble in many ways those recognised as symptoms of anaphylaxis."

Here are interesting and illustrative cases of hypersensitivity to certain common foods. One recorded by Talbot (1916) deals with milk. Reactions due to proteins in this food are fairly common. A healthy baby, which was breast-fed till it was 8½ months old, was given cow's milk and barley water without any ill-effects. This was stopped for a few weeks, but when cow's undiluted milk was added to the diet the child vomited and showed decided symptoms of illness and within an hour its body was covered with an urticarial rash. Substitution of goat's milk for the cow's milk at once stopped the trouble.

Cases of hypersensitiveness to egg albumen frequently occur and several typical instances have been recorded. Coues (1912) described a case where a child about one year old was given the white of an egg which immediately caused nausea and vomiting. About 8 months later the child was again given white of egg. Violent sneezing and all the symptoms of an acute cold in the head followed, an extensive urticarial rash appeared on the body and the eyelids became œdematous. The temperature remained normal and there was no marked prostration.

Jordan (1931) in reference to sensitisation to egg albumen states that in some cases the amount of the specific protein that suffices to produce the reaction is exceedingly small. One physician writes of a patient who " was unable to take the smallest amount of egg in any form. If a spoon was used to beat eggs and then to stir his coffee, he became very much nauseated and vomited violently."

Kennedy (1936) recorded a peculiar case of allergy in a woman, caused through the consumption of chocolate. She had recurring eczema on various parts of the body but was otherwise healthy. When put on a special diet the eczema completely disappeared within a fortnight. Later, however, the disease recurred and strict inquiry revealed that she had eaten chocolate. She heeded a

warning not to do so again and her skin got quite well and she was able to take all foods. A piece of chocolate was then given as a test with the result that the eczema reappeared.

Lefevre (1930) described a case of illness in a soldier which may have been due to sensitisation. After eating pineapple he showed symptoms of vomiting, pain in the stomach and finally lost consciousness. Two others who partook of some of the same pineapple were not affected. This type of food idiosyncrasy has been described on several occasions. McBride and Schorer (1916) collected particulars of 60 cases of food sensitiveness causing skin trouble, such as urticaria and erythema. Fish, tomatoes, cheese and eggs were among the foods causing urticaria, and cereals and pork the erythema, the illness appearing within less than 4 hours after consumption of the food. Tomatoes and cereals generally produced these conditions in less than 1 hour, the eruption lasting from 1 to 12 hours and in a small percentage of cases from one day to one week.

With regard to the incidence of food allergy, the only definite figures available are the result of a questionnaire issued to 400 students and nurses as to the presence of allergy in their personal and family histories. The result showed probable food allergy in more than 30 per cent. of all persons.

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# PART III

## CHAPTER XII

## BOTULISM [1]

### Historical

This comparatively rare but extremely interesting type of food poisoning apparently had its origin on the Continent. According to historical records, 'sausage poisoning,' as it was then termed, was prevalent in Germany as long ago as 1735, and was at first believed to be due to contamination of the sausages by the copper and lead vessels in which they were prepared (Müller, 1735–93) and Kerner (1755–89).

The earliest recorded outbreak which attracted the attention of the medical profession occurred in Wildbad, near Würtemburg, in 1793 (Müller), and affected 13 persons, 6 of whom died as a result of eating 'schweinsmagen' or 'blünzen' (blood puddings or visceral sausages).

The characteristics of the fatal nature of the illness were brought to the notice of the Court Physician (Keiser), who suspected belladonna poisoning, owing to the symptoms somewhat resembling those caused by this vegetable poison. At Hofe Mosburg, in 1799, an outbreak occurred involving 5 persons, 2 of whom succumbed. The son of the family was accused of mixing henbane seeds with the sausages with criminal intent.

Subsequent outbreaks caused the authorities to realise how unsatisfactory were the methods of preparing cheap meat foods. In 1802 Jaeger published an official warning from Stuttgart pointing out the dangers arising from the consumption of unwholesome sausages and other 'made-up' meat foodstuffs and issued instructions for their proper manufacture. He suggested that the toxic action of the sausages must have been due to the presence of some vegetable seeds or spices and not to any mineral poison. In spite of the warning, however, sausage-poisoning increased in Würtemburg, and cases, some fatal, occurred in parts of Southern Germany. Ostertag (1907) comments upon this distribution: "If we ask why botulism occurs so frequently and causes so many

[1] Botulismus (from Latin *botulus*, a sausage), sometimes known as Allantiasis, Ichthyosismus or Würstvergiftung.

deaths in Würtemburg, an explanation is to be found, in the first place, in the great development of sausage manufacture, and in the consumption of sausages in Würtemburg, and, also, in the ignorance previously exhibited in preparing certain kinds of sausage as 'leberwürste' and 'blutwürste,' for consumption at a considerably later date. I emphasise the word 'previously' for the gradually diminishing number of cases of sausage poisoning in the last decades proves that a change has taken place in this regard. In Northern Germany, on the other side of the Main, it is the custom to eat sausages prepared from the viscera, as, for example, 'leberwürste' and 'lungenwürste,' only in a fresh condition. At any rate, smoked 'leberwürste' in Northern Germany is exceedingly rare, except in Thuringen. The so-called long-keeping sausages of Northern Germany, which are the only kinds which are preserved for the period of months or one year, consists of musculature, which, when properly conserved, resists decomposition much longer than lungs, liver or blood. In the etiology of sausage poisoning in Würtemburg, however, smoked visceral sausages play an important rôle. These sausages are poorly adapted for keeping for a long time, since they contain material which spoils readily."

During the years 1820–22, Justinus Kerner, a noted physician, made important and systematic investigations into the cause of sausage poisoning and carried out numerous experiments to prove his theories. Later, he published two monographs in which he related the history of the malady and referred to epidemics in other parts of Germany. Subsequently, laws were enacted and the disease was made notifiable. Records reveal that during the period 1735–1874, 920 cases with 366 deaths occurred in Würtemburg (Meyer, 1928). In Northern Germany the malady was comparatively rare. Nevertheless, in 1822 and 1828 the Royal Imperial Government at Arnsberg issued public warnings against the consumption of semi-solid, sour and malodorous sausage.

Leighton (1923) remarks : "Altogether there would appear to have been about 1200 cases of botulism in Germany during the past 130 years, with a mortality of 360, or 30 per cent."

From time to time cases of sausage poisoning, however, continued to be reported from Anhalt, Baden, Bavaria, Hessen, Holstein, Prussia, Pomerania, Posen, Saxony and in the Provinces of Hanover and Silesia. Records also show that the malady was present at times in Austria, Denmark, Holland, Hungary, Russia and England.

With regard to the disease in England, it may be of interest to note in passing that John Tribe (1860), Medical Officer of Health for Hackney, London, mentions cases of sausage poisoning. He remarks: "Medical literature in this country contains but few records of cases caused by diseased or putrefying meat." He quotes from Taylor's work on medical jurisprudence. Taylor, when discussing poisoning by cheese and sausages, observed: "Although these articles of food have frequently given rise to symptoms of poisoning in Germany, there is, I believe, no instance of their having proved fatal in England." Later in his work, however, he gives an account of three fatal cases of poisoning from eating pig's liver sausages.

The majority of the earlier outbreaks of sausage poisoning in Central Europe was caused by the consumption of certain prepared meat foods, such as blood sausages, 'blütwurste' (Jaeger, 1802); liver sausage, 'leberwürste' (Von Autenrieth, 1815); 'schlackwürst,' made from pork, veal and calf blood, and 'presskopf' prepared from livers or tongues and hog's heads (Horn, 1830). These popular foods amongst the poorer classes, were often eaten raw or partly cooked and sometimes in a semi-decomposed condition. Scientific observers naturally supposed that such foods were the primary cause of the disease, and for many years their investigations and experiments were based on this supposition. As time went on, however, observations proved that similar symptoms and illness were produced by the ingestion of many kind of foods other than spoiled sausages. They included smoked pork (Hauff, 1829), cheese, fats and ham. The latter foodstuff was responsible for a large number of cases of poisoning recorded by observers over a considerable period.

Von Autenreith (1833–5) drew attention to the similarity of fish poisoning in Russia to sausage poisoning in Würtemburg. Many such outbreaks of 'fisckyergiftung' in Russia (Jaechnichen, 1850), Schlossberger (1852) and elsewhere (Bohm, 1876) were reported from time to time and caused much discussion. Proof was forthcoming through Madsen (1912) who, in investigating an outbreak of fish poisoning in Oro, Denmark, isolated a bacillus identical with B. botulinus. The patients exhibited the characteristic symptoms of botulism after the ingestion of a pickled mackerel. One of the cases terminated fatally. The fish had a rancid odour, and the toxin in the brine was neutralised by the antitoxin prepared against the bacillus isolated by Van Ermengem at Eezelles.

Innumerable theories were advanced from time to time to

account for the poisonous nature of the incriminated foods; these included chemical poisons, putrefactive alkaloids, ptomaines, ferments, vegetable organisms, moulds and other low forms of life.

As a result of constant experimental work by Kerner, it was established that the poison was developed within the sausage and was not caused by outside agents. It is a significant fact that Kerner (1824) came to the conclusion that the exclusion of air from the sausage was necessary for the production of the poison, and that sausages in large casings (incompletely filled) made smoking difficult and were more poisonous than meat sausages enclosed in small casings. This was confirmed by other investigators. Kerner concluded that the odour was not that of ordinary putrefaction. The taste was described as sour, bitter and burning. He suggested that cooking the sausage might inhibit the action of the poison.

Schlossberger (1852) noted that meat sausages, which were expensive and were consumed by the wealthier classes, were usually packed in small casings and prepared under cleaner and better conditions and rarely poisonous. He also observed that the poisonous sausage had a peculiar cheese-like odour.

Later, as a result of experiments, Cormack and Corneliani (1852) demonstrated that during the process of smoking the sausages, a poisonous acid (pyroligneous) was given off when burning certain woods. This acid, they believed, accumulated in the sausages and caused poisoning.

Emmert and Kuhn (1824) suggested that the fatal cases might be caused by the formation of prussic acid, owing to the blue-black colour of the blood found in the bodies of persons who had died from sausage poisoning. This theory, which was contested by other workers, caused Kerner to carry out further experiments with the result that he found the symptoms produced by prussic acid were quite unlike those caused by sausage poisoning. Moreover, prussic acid could not be detected in the blood or tissues from the fatal cases.

In the course of his many researches in the hope of finding the toxin substance, Kerner isolated a fatty acid substance from decomposed sausages and rancid fat which he termed 'leichensaüre' and believed this to be the true toxic agent, as it produced symptoms of sausage poisoning in animals, similar to those seen in the human cases.

Buchner (1823), Kastner (1823) and Horne (1828), Dann (1828) and other workers, carried out a series of investigations and

experiments with fats and acids but came to no satisfactory conclusions, and Kerner's theory was not confirmed.

Bodenmüller (1834) and Krugelstein (1839), after studying the subject, were of the opinion that sausage poisoning was not due to winter-prepared unsmoked 'leberwürste,' but that the poison was produced by the methods commonly employed in Würtemburg in smoking and heating the sausages. Liebig (1843) believed that the disease was due to the poisonous action of a ferment. Schlossberger (1852), however, was not in agreement with this, and suggested that the disease was caused by the presence of certain organic bases of the alkaloidal group.

Heller (1853) was the first to suggest that a microscopic vegetable mould inside the sausage was responsible for the formation of the poison, whilst other investigators (Van den Corput, 1855, Wittig, 1856, and Kasper, 1858) opined that moulds, algæ (Sarcina botulina), was the probable cause of the illness, but no conclusive evidence was forthcoming. In 1886 Von Aurep advanced a theory that sausage poisoning was caused by a putrefactive base (ptomatoatropin) which he obtained from tainted fish. In the same year Ehrenberg isolated several putrefactive amines from a poisonous sausage, two of which he believed were responsible for the poisoning. He failed, however, to produce the characteristic symptoms in experimental animals.

Nauwerck (1886) gave it as his opinion that the substances described by Ehrenberg were of bacterial origin and isolated three bacilli from the identical sausage, one of which liquefied gelatine and caused putrefaction of sterile blood. Later, Redner isolated a similar bacillus from the intestines of a hog. Nauwerck concluded that this organism was ingested with the food and caused putrefaction of the intestines, resulting in auto-intoxication which produced sausage-poisoning symptoms.

In spite of the numerous theories put forward from time to time and the vast number of experiments carried out by research workers, scientists and members of the medical profession, the primary cause of this form of food poisoning was undiscovered. It was not until December, 1895, that the responsible organism was isolated, and described by the Belgian scientist Émile Pierre Marie Van Ermengem of Ghent, during the investigation of an outbreak which occurred among the members of a musical society who were engaged to perform a dirge at a funeral in the village of Ellezelles (Hainault). After the ceremony the members partook of a cold repast in which a pickled ham played a prominent part.

Thirty-four of the members were taken ill and 3 died. The incriminated ham, which had been pickled in brine for 4 months, had a musty rancid odour and bitter taste and was pale and partially discoloured but not decomposed. It came originally from a pig slaughtered in the previous August, and in the fresh state a part had been consumed without ill-effects. Van Ermengem isolated a large anaerobic bacillus from the remains of the ham and from the spleen and contents of the large intestine from one of the fatal cases. By cultivating the bacillus under anaerobic conditions he ascertained that a powerful and deadly toxin was manufactured in the surrounding medium. He made watery extracts from the remains of the ham and injected it into experimental animals and produced in them the characteristic neuro-paralytic signs observed in the fatal human cases. The most typical symptoms were seen in cats.

This remarkable discovery thus proved the casual relationship of the bacillus and its toxin to the disease, and at the same time put an end to all speculations as to the nature of the poison causing the symptoms seen in the previous cases.

Van Ermengem compiled a complete list of the characteristic symptoms of the disease culled from close observation of the patients. He named the organism Bacillus botulinus, and the disease became known as Botulism. Van Ermengem, as a result of his investigations, which were confirmed by other observers, came to the conclusion that botulism was not an infection but an intoxication, the bacillus not growing in the human body.

Kemper (1897), by means of Van Ermengem's cultures, showed that the botulinus toxin causes the development of a powerful specific anti-toxin in the body of goats.

Van Ermengem (1906) isolated another strain of B. botulinus during an outbreak amongst 12 persons at Iseghain in West Flanders.

In 1900 Römer investigated an outbreak in the district of Alsfeld, Germany, due to the consumption of a pickled ham, which caused the illness of 4 persons. He isolated a bacillus similar to the one discovered by Van Ermengem.

Van Ermengem's finding was confirmed by Landmann (1904), Ornstein (1913), Schumacher (1913) and other observers. For some years after the discovery of B. botulinus, the disease of botulism was looked upon as rare and of academic rather than of practical importance. It was presumed that a meat product was essential for the satisfactory growth of the bacillus. In 1904,

however, an outbreak of illness, described by Fischer (1906), occurred at the Alice Cooking School in Darmstadt amongst 21 persons, 11 of whom died within 4 to 5 days after the consumption of a cold home-canned white bean salad. The contents of the can had a rancid odour, but the beans showed little disintegration. A bacillus identical with B. botulinus was isolated, which yielded a toxin fatal to guinea-pigs in doses of 0·0003 c.c.

This was a typical example of an outbreak of botulism, not of meat origin, and doubtless attracted the attention of observers to the possibility of canned vegetables being suitable media for the growth of the bacillus and the deposition of the toxin.

Modern history records that botulism has been prevalent during the past two or three decades in the United States of America. Dickson (1917) considered that the disease was more common than is shown by the records.

In 1918, however, he remarked : " A review of the American literature reveals that very few cases of botulism have been recognised in this country, but in a survey of the available cases of food poisoning during the past 25 years it was found that there have been a number of cases in which the symptoms are more or less indicative of this condition."

Dickson (1918) quotes outbreaks recorded by Jellinck (1902), Sheppard (1907), Peck (1910), Stiles (1913), Wilbur and Ophüls (1914), Frost (1915), Lancaster (1916), Curfman (1917).

In America botulism has been mostly associated with the consumption of canned or preserved vegetables and fruits. ' Home-canning ' is carried out to a large extent, and this, especially as regards fruits and vegetables, is the real explanation of the relative frequency of the disease in that country.

Dickson (1917) carried out experiments to test the efficacy of the ' cold-pack ' method usually employed in home-canning, where the filled jars are heated in a wash boiler to a temperature of 212° F. (100° C.) for 120 to 180 minutes. Dickson concluded that the ' cold-pack ' was not efficient if the raw vegetables had been contaminated with the spores of B. botulinus.

Botulism apparently increased in the United States during the period 1910–22, but later showed a distinct tendency to decrease. Meyer holds the opinion that the decline in the number of outbreaks of single cases is attributable to energetic preventive measures, both educational and legal.

Jordan (1931) says : " In the United States searching epidemiological and bibliographical inquiries by Meyer and his

associates have brought to light the probable occurrence of about 550 authentic cases of botulism in the period 1899–1927. It cannot be determined what proportion of cases escape observation and record. For various reasons part of the particular period covered by these figures (1918–25) seems to have had an unusually high ratio of botulism outbreaks. In 1926 there were only 6 cases (3 outbreaks) and in 1927 only 11 cases (5 outbreaks) in the United States."

Botulism has been systematically studied by numerous scientific investigators in the U.S.A., and many brilliant contributions have been added to the literature on the subject by Bengtson, Dack, Damon, Dickson, Dubovsky, Easton, Esty, Geiger, Jordan, Meyer, Ophüls, Tanner, Thom, Wilbur and others.

Dickson (1918), as a result of his intensive studies and investigations, compiled a clinical and experimental study on botulism. It may be of interest in passing to append some of his conclusions :

" 1. Botulism is endemic in the U.S. and is comparatively common in the Pacific Coast States.

" 2. It is not essentially a meat poison but may also occur in canned vegetables and fruits.

" 3. The methods which are usually employed in the home-canning of vegetables and fruits are unsafe.

" 4. All home-canned vegetables should be cooked before they are eaten.

" 5. Botulism is a frequent cause of the so-called ' limber-neck ' of domestic fowl, and it may be responsible for certain types of paralysis of various kinds of domestic animals, including dogs.

" 6. The occurrence of limber-neck in domestic fowl, if it has developed after they have eaten refuse from the kitchen, may be an indication for the prophylactic administration of the botulinus antitoxin to all persons who have eaten the suspected foods."

In 1919 several spectacular outbreaks were caused by the consumption of factory-preserved olives, and for the first time the canning industry in America was confronted with the fact that Cl. botulinum was a real instead of merely a potential danger.

Impressed by the grave responsibility thrust upon the canning industry, the National Canners' Association, the Canners' League of California, and the California Olive Association proposed an investigation to determine the danger of botulism, how it arose and how it could be avoided and overcome. A Commission (Geiger, Dickson and Meyer) was formed in California and as a result of

their researches " The Epidemiology of Botulism " was published in September 1922 by direction of the Surgeon-General.

Since 1929 the entire preserved foods industry of California has been controlled through legislation by the California State Department of Public Health, and revised tentative regulations covering sterilisation of products are issued during the packing season of the year for the guidance of the industry.

Among the principal works in the United States of America containing matter relating to botulism are those by Damon (1928), Jordan (1931), Meyer (1928), Bengtson (1924), Strader (1939), Tanner (1933), Thom and Hunter (1924). In addition, and as a result of experimental study of the disease by various workers, numerous articles have appeared from time to time in medical and scientific journals.

The presence of botulism in Russia (ichthyism) was reported in 1927 by Zlatogoroff and Soloviev. Twelve outbreaks occurred between 1881 and 1926 with 52 cases and 35 deaths. The disease appears to be endemic in the Republics.

In the Great War (1914–18) no cases of the disease were reported amongst the British or Allied troops, although the consumption of canned and preserved foods was enormous. This was doubtless due to rigorous inspection and supervision of supplies and to increased efficiency in the canning industry.

Dorendorf, however, recorded 5 cases of disease as occurring in the German Army, and Bitter observed 3 outbreaks at Kiel in 1918–19.

## Botulism in Great Britain

No authentic cases of botulism were recorded in Great Britain until August 1922, when an outbreak (known as the Loch Maree tragedy) occurred at Loch Maree, Gairloch, in the Western Highlands of Scotland. Eight persons were affected, and all died within a week after eating sandwiches made with wild duck paste. The outbreak formed the subject of a special report to the Scottish Board of Health by Gerald Leighton (1923). As a result of this lamentable tragedy the Ministry of Health made arrangements for a supply of botulinus antitoxic serum to be available at several centres in England and Wales and issued instructions for its use.

Leighton (1923) published his work on " Botulism and Food Preservation " and included a comprehensive study of the Loch Maree outbreak. This publication marked an epoch in the medical literature, as it was the first British work on the subject.

In the same year Humphrey Milford contributed an up-to-date review on the disease in the *Medical Science Extracts and Reviews*.

The Medical Research Council in 1929 published " A System of Bacteriology in Relation to Medicine " in which Cl. botulinum was dealt with by Hewlett, O'Brien and Bullock.

Since the Loch Maree outbreak in 1922 there were no authentic cases until August 1935, when 3 deaths definitely due to botulism occurred in North London. The Chief Medical Officer, Ministry of Health (1935), remarks : " These fatal cases were all adult women, and there were two others, both male, in which the same intoxication was almost certainly a contributory cause of death . . . one man, who had partaken of the dish responsible for two of the fatal cases, recovered after presenting slight symptoms of botulism and being treated with botulinus antitoxin."

Later, in August 1935, another fatal case (a man) occurred in London, in which the symptoms suggested botulism. The findings at the autopsy were consistent with death from botulism. No further cases of botulism have occurred in Great Britain since 1935.

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# SYMPTOMATOLOGY

## Symptoms

The symptoms of botulism which are manifested in man as a result of the gradual absorption of the toxin produced by Cl. botulinum, are of a peculiar and characteristic nature. They differ markedly from those observed in other types of food poisoning and vary in severity and duration in different outbreaks.

Van Ermengem (1897) remarks : " The symptoms of botulism are so uniform and true to nature that for the recognition of the disease clinical appearances are alone sufficient. The picture is mainly made up of neuroparalytic disturbances of central origin. These processes find their expression in certain changes in the secretory functions of the digestive tract and in symmetric, generalised or localised motor paralyses caused by lesions seated in the ganglion cells of the bulbar and spinal nuclei."

In typical cases the symptoms develop in the same sequence, and the whole illness usually lasts from 36 hours to 5 days, but may be prolonged to a week. The majority terminate fatally, being due to cardiac or respiratory failure. In some instances, death may take place within 24 hours from time of onset. Few recoveries are recorded and in such cases convalescence is very prolonged.

In a series of 173 in America, 18 patients died within 48 hours after the food was eaten and one survived for 26 days ; but 117 persons, or 67·7 per cent., died in from 3 to 6 days after ingesting the poison (Geiger, Dickson and Meyer 1922).

Muller (1870) recorded that of 150 fatal cases, the majority died in from 4 to 8 days after the poisoning, and he added that few persons died who survived for more than 10 days.

The symptoms usually commence anything from 12 to 36 hours after ingestion of the toxic material, but may be delayed 2 to 3 days or even longer, considerable variation occurring in individual cases. Only rarely do they appear earlier than 12 hours. To illustrate this, Dickson (1918) says : " In a series of over 200 cases a few occurred within 12 hours, 74 per cent. within 48 hours, and all but 8 cases within 4 days after the poisonous food was eaten ; 4 victims, however, first showed symptoms on the 5th day, 3 on the 6th day and 1 on the 8th day."

In a few instances the true intoxication manifestations may be preceded by gastro-intestinal disturbances, as observed in ordinary types of food poisoning. The earliest observers noted the gastro-intestinal disturbances, and Dann (1828) has suggested a division of the symptoms into two groups, i.e. (*a*) the ' irritative ' group and (*b*) the ' paralytic ' group; this was not generally accepted. Schlossberger (1852) and Muller (1870) pointed out that the irritative group of symptoms was frequently absent.

According to Geiger, Dickson and Meyer (1922), approximately one-third of the cases of botulism have exhibited disturbances which usually come on early and apparently as a result of irritation of the alimentary tract by the spoiled food ingested. The remaining two-thirds show the typical symptoms of intoxication immediately following the incubation period.

In the classical outbreak of botulism at Loch Maree in Scotland (1922) intestinal disturbances were slight or absent altogether.

Regarding the incubation period, which is usually under 24 hours, Leighton (1923) remarks: "The most unfortunate thing about the symptoms is that after the patients have taken the poison into their system, and while it is being absorbed and carried to the brain, there is a period extending over some hours during which no symptoms appear at all. The patient is quite unaware of what has taken place."

The first signs are usually a peculiar feeling of lassitude, fatigue, headache and dizziness, sometimes accompanied by progressive and definite weakness of the muscles of the arms and legs; vertigo is not uncommon. When gastro-intestinal disturbance is present there may be nausea and vomiting of a yellow colour, bitter taste and irritating with a feeling of weight or actual pain in the region of the stomach. Diarrhœa may persist for a few hours or longer. As a rule intestinal disturbance is absent or of a transitory nature and of secondary importance. Persistent constipation (paralysis of the muscles of the wall of the intestines), which is a distinct feature of botulism, may be the initial symptom, or immediately follow the diarrhœal stage and may or may not be accompanied by retention of the urine.

As the central nervous system becomes involved, which Bronfenbrenner and Schlesinger (1924) concluded was the result of the absorption of the toxin through the mucosa upper intestinal canal—although according to Dickson (1918) instances have been recorded in which persons were poisoned by tasting very small amounts of the poisonous food and not swallowing any of the toxin

—the visual disturbances begin to make their appearance, i.e. dimness or blurring of vision, double vision (diplopia), and early involvement of the third cranial nerve (ptosis), with drooping of the eyelids. The pupil of the eye increases in size (mydriasis) and involuntary oscillation of the eyeball (mystagmus) is not uncommon ; fixation in the socket sometimes takes place. There is loss of reflex to light stimulation and finally complete loss of accommodation.

The ocular symptoms—impairment of the extrinsic and intrinsic muscles of the eye—are highly characteristic of the fatal form of intoxication and may be the first serious signs of the disease. Dickson (1918) points out that a fairly large number of cases of botulism are first seen by ophthalmologists and opticians. As intoxication proceeds, speech becomes difficult (husky voice), indistinct (dysphasia) and eventually loss of voice (aphonia) occurs. There is a sensation of suffocation and constriction in the throat, owing to paralysis of the pharyngeal and laryngeal muscles. The tongue becomes sluggish in movement, is heavily coated, increases in size and may become paralysed. Swallowing is difficult (dysphagia) and attacks of strangling occur when an attempt is made to swallow food. During these attacks, regurgitation of fluids through the nose sometimes takes place. The muscles of the face and neck become affected, giving the patient a pale, dull and mask-like expression.

In the early stages of the illness, restlessness, insomnia, irritability and sometimes hysterical attacks are observed as a result of the patient not being able to make himself or herself understood (except by writing) or to swallow. Secretory disturbances are most marked. In some cases there is unnatural dryness of the mouth, throat and nose, the mucous membrane shrinks, shrivels and desquamation may occur. In others, a thick glairy mucous exudes and stretches across the throat and pharynx, resulting in a croupy cough when efforts are made to free the mucous from the pharynx. Sweating may be absent, the skin on the palms of the hands and soles of the feet becoming dry and thick, but if present, is profuse and offensive. Partial deafness may ensue.

In mild attacks of botulism there is inco-ordination of the muscular movements of the arms and legs.

Extreme general muscular weakness is a marked feature of the illness, the patient being unable to raise the head, arms or legs.

Among the other characteristics are absence of sensory disturbances, pain, consciousness and mentality unimpaired through

the whole course of the disease. The temperature is sub-normal ranging from 96° to 98° F. Pulse-rate, which is comparatively slow in the early stages (50 to 60 per minute), later becomes rapid and as high as 100 to 150 per minute. This combination of rapid pulse-rate and sub-normal temperature is a striking feature of the illness. In the early stages respiration is not interfered with but eventually breathing becomes irregular, shallow, rapid and difficult. The normal blood-pressure and the skeleton muscle reflexes are intact. The patient becomes gradually weaker, the intercostal muscles are fatigued, the exhaustive strangling spells are more frequent, and finally death occurs from paralysis of the respiratory centres and cardiac failure, consciousness remaining almost to the end.

During the last few hours of the illness, broncho-pneumonia may supervene, which causes some fever with a consequent rise in temperature.

Cases have been recorded where coma has set in before death. According to Geiger, Dickson and Meyer (1922): "It has been frequently observed that the heart continued to beat for several hours after voluntary respiration had ceased, and cases are recorded when artificial respiration has maintained life for several hours after voluntary respiration had ceased. Usually there is terminal asphyxia with cyanosis, and occasionally the patient dies in a strangling spell. It is not uncommon that there may be apparent improvement in the general condition of the patient but that death results from insufflation broncho-pneumonia."

As to the cases which recover, it is generally recognised that the intoxication usually reaches its maximum in from 4 to 8 days, then it begins to subside. If, after 10 days, the patient survives, improvement usually follows, but convalescence is extremely slow and tedious. Recovery of speech and swallowing (strangling) takes place early. Owing to digestive troubles, the patient is thin and emaciated. Muscular weakness, vertigo and constipation may persist for months and the visual disturbances are the last to clear up. Complete recovery, however, takes place, although it is some considerable time before the former condition of health is attained.

## Mortality

The mortality rate for botulism varies considerably in individual outbreaks, and figures ranging from 20 to 87 per cent. have been recorded from time to time by various observers. In

Differential Diagnosis between Botulism and the other kinds of Food Poisoning

| | Botulism. | Food Poisoning. |
|---|---|---|
| Incubation period . . | 12–36 hours, sometimes delayed to 48 hours or even longer. | 6–12 hours. Short usually. May be only half an hour or delayed to 30 hours. |
| Onset . . | Gradual. | Sudden. |
| Gastro-intestinal symptoms . | Frequently absent; if present, slight and transitory. | Early and marked, tongue heavily coated, foul breath. |
| Vomiting . | When gastro-intestinal disturbance is present. | Very common. |
| Diarrhœa . | May or may not be present. Obstinate constipation early: may be the first symptom. | Usually severe. Offensive motions which later may become green and watery. |
| Abdominal pain | May be present. | Marked, often severe. |
| Muscular cramps | Absent. | Common. |
| Temperature . | Sub-normal 96° to 98° F. | Elevated at first. |
| Prostration . | Gradual and late. | Marked and early. Persistent into convalescence. |
| Rashes . . | Absent. | Herpes common, as also erythematous and urticarial rashes followed by desquamation. |
| Nervous system | Nervous symptoms from commencement of illness. Disturbances of vision, generally the first symptoms noticeable. Paralysis of accommodation, diplopia, mydriasis, ptosis, and internal strabismus, aphonia, diuresis or anuria and peresis of tongue are also common. | Nervous symptoms only appear in the later stages of the illness and following the acute gastro-intestinal symptoms. |
| Duration of symptoms . | Protracted and progressive. Whole illness usually lasts 36 hours to 5 days but may be prolonged to a week. | Acute symptoms diminish rapidly after 48 hours to 3 days, with exception of prostration. |
| Mortality . | 30–70 per cent. or higher. | 1–2 per cent. generally, but varies. |

the Loch Maree outbreak in Scotland (1922) it reached 100 per cent. The rate in the U.S.A. is much higher than in Europe.

Regarding the death-rate among cases in the early history of botulism, Kerner (1820–22) records a series of 159 cases with 84 deaths—a mortality rate of 52·8 per cent.

In Schlossberger's (1852) series of 400 cases, there were 150 deaths, a rate of 37·5 per cent.

Dickson (1918) summarises the early figures for cases and deaths in Germany from official sources obtained by Meyer, showing that the disease was still comparatively frequent in that country :

| *Date.* | *Cases.* | *Fatal.* |
|---|---|---|
| 1793–1820 . . . . | 76 | 37 |
| 1820–1822 . . . . | 98 | 34 |
| 1822–1886 . . . . | 238 | 94 |

Since 1886 there have been about 800, about 200 of which were fatal.

In the United States, Geiger, Dickson and Meyer (1922) collected data on 91 outbreaks up to 1922 with a total of 345 cases of which 213 were fatal, a case mortality rate of 61·7 per cent.

In the 11 recorded outbreaks in the Colorado State (1912–18), summarised by Hall and Gilbert (1929), the death-rate was 71·7 per cent.

In Germany the death-rate averaged 25 per cent. (Meyer, 1913) and the United States 65 to 70 per cent. (Burke *et al.*, 1921).

According to Zlatogoroff and Soloviev (1927) the fish-poisoning outbreaks of botulism in Russia were accompanied by a high mortality amounting to about 67·3 per cent.

The difference in the rate in America and Europe is probably attributable to the nature of the particular kinds of food consumed in the country concerned. The mortality rate for children is higher than for adults.

Apparently the earlier the symptoms of the disease, the higher the mortality rate. In America, Burke (1921) reported that the death-rate among those showing symptoms in 24 hours, 84 per cent. died ; of those that developed symptoms in 72 hours, 55 per cent. died, and of those alive after the eighth day, 20 per cent. died.

Geiger, Dickson and Meyer (1922) state : " In a series of 246 cases where data were available and of which 173 resulted fatally, it was found that 147, or 85 per cent., of the fatal cases were persons in whom the onset of symptoms occurred within 48 hours

after ingestion of the poison. In many outbreaks there is indication that the time of onset of symptoms is directly dependent upon the amount of poison ingested, and this observation shows that the severity of the illness and the mortality rate is also directly dependent upon the same factor."

From the above figure, it will be seen that the mortality rate for botulism is very high compared with other types of food poisoning.

Meyer (quoted by Jordan, 1931) points out the interesting fact that isolated cases and deaths from botulism occur most frequently in women who, as cooks and housewives, are likely to taste foods of their own preparation, which, from odour or appearance, they suspect.

## Climatic Influence, Seasonal Prevalence and Intoxication Rate

Botulism bears no relation to climate. As a rule the disease is usually associated with preserved foods, the consumption of which generally takes place in the winter months, when fresh food is not obtainable.

According to Dickson, Geiger and Meyer (1922) more than half of all the outbreaks in California occurred between October to February in contrast with bacterial food infections, which are usually prevalent during the summer months.

The intoxication rate is very high (100 per cent.). As a rule all who consume the toxic food become ill. Uneven distribution of the toxin in food is possible, but extremely rare.

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## CHAPTER XIV

# CAUSATION

BOTULISM is an example of food poisoning due to bacterial products formed outside the human body. It is an intoxication and not an infection. In other words, for human botulism to result, the causative organism must multiply and produce its toxin in the food before it is consumed.

## Bacteriology

Bacillus botulinus was described by Van Ermengem in 1896 as a very large slightly mobile anaerobic bacillus with rounded ends, 4–6 microns in length and 0·9–1·2 microns in breadth (a micron is 0·000039 of an inch). It sometimes occurred in pairs but rarely in filaments. The spores were terminal and somewhat wider than the bacillus, giving it a club-shaped appearance, and resisted ordinary stains. They were destroyed when exposed to 80° C. for 30 minutes. Spindle forms were sometimes observed. The bacillus was strictly saprophytic and would not produce its toxin in the animal body. It may truly be called a pathogenic saprophytic and placed in the same category as atropa belladonna (deadly nightshade) and nux vomica, amongst the green plants.

The organism, which had four to eight very fine flagella of wavy form, was gram-positive, but decolorised rapidly when treated with alcohol. Characteristically round, transparent yellowish-brown colonies were formed on glucose gelatine medium, containing granular bodies in motion. Gelatine was liquefied but milk was not changed or coagulated. Gas was formed in glucose broth or agar but in broths containing lactose or saccharose no gas was produced. A butyric acid (slightly rancid) odour was emitted during cultivation in various media. Van Ermengem believed that the amount of gas formation was an indication of the activity of the bacillus, especially in toxin production.

Media which gave an acid reaction to litmus or phenolphthalein prevented growth. Good growth, however, was obtained in a slightly alkaline medium containing 1·43 per cent. of $Na_2CO_3$ incubated at a temperature between 20° and 30° C. (optimum temperature), producing a powerful toxin which when fed to guinea-pigs and mice proved very toxic. Rabbits, rats, pigeons,

dogs, hens and cats withstood large doses of the toxin. Cats on subcutaneous injection showed all the characteristic symptoms of botulism. Monkeys (rhesus) were susceptible both to subcutaneous inoculation and to feeding. Frogs and fish were refractory. (After Bengtson, 1924.)

Although a strict anaerobe, the bacillus may be cultivated under imperfect anaerobic conditions, if in symbiosis with certain aerobic bacteria, with the white Sarcina or with B. subtilis (Römer, 1900), and according to Harrass (1906) and Tarozzi (1905), will grow in freshly prepared bouillon conditions if a piece of sterile flesh or potato is placed at the bottom of the culture tube (Dickson, 1918).

A small amount of sodium chloride, 0·5 per cent., is necessary for the growth of the bacillus, but too much will inhibit development. Van Ermengem (1897) found that 2 per cent. sodium chloride was deleterious to the growth of B. botulinus in bouillon. Growth is stopped by 6 per cent. sodium chloride ; consequently meat pickled in brine containing more than 6 per cent. will not become contaminated with the toxin.

Since Van Ermengem's discovery of B. botulinus several strains of the organism have been isolated from time to time in Europe. They differ in their characteristics and serological reactions from each other and from the original B. botulinus which of course has now died out. Damon (1928) states : " Of the numerous strains now extant, that described as Lister No. 94, in the publications of the Medical Research Committee of Great Britain most nearly approaches the characteristics of Van Ermengem's culture and with this the American types may be compared."

A slight difference in regard to the degree of mobility may be noted. Van Ermengem states that his culture was very slightly motile. The Lister culture is described as actively motile (Bengtson, 1924).

## Clostridium botulinum

The two main toxigenic types, designated ' A ' and ' B ' respectively, caused botulism in man. A and B were suggested by Burke (U.S.A. 1919), to distinguish the two groups. Type A, which is much commoner than Type B, has been isolated frequently from cases of botulism on the Pacific States of America, and Type B from cases occurring in the Eastern States and in Europe. In 23 outbreaks recently in America, 19 were due to

Type A and only 4 to Type B (Topley and Wilson, 1936). A third. Type 'C,' was isolated by Bengtson (1922–3) from the larvæ of Lucilia cæsar, one of the green-bottle flies, and is associated with a disease termed 'limberneck' of chicken, caused by the ingestion of the larvæ (Wilkins and Dutcher, 1920, and confirmed by Graham and Boughton, 1923), and ducks in the United States of America and other countries (Gunnison and Coleman, 1932).

Further types were isolated in Australia and South Africa. An epizootic paralytic disease of cattle known as 'Midland cattle disease' exists in Tasmania, which was investigated by Seddon (1922). He isolated a bacillus which he named B. parabotulinus. This organism is considered to be a member of the Type C group.

A fourth, Type 'D,' has been associated with a disease of horses and cattle, 'lamziekte,' in South Africa and isolated by Theiler and Robinson (1926–7), Robinson (1929), Gunnison and Meyer (1929).

A fifth, Type 'E,' was demonstrated by Theiler and others (1926–7), Theiler (1928), in horses in South Africa.

Regarding the types of Cl. botulinum isolated from botulism in animals, birds, etc., Topley and Wilson (1936) remark: "Great confusion exists about the exact identity of the various organisms isolated. Because some of them differ in their toxin production from the classical *Cl. botulinum* A or B types, the names *Cl. parabotulinum*, *Cl. parabotulinum bovis*, or *Cl. parabotulinum equi* have been suggested, and the disease caused by them has been termed parabotulism. This is not the place to discuss bacteriological nomenclature, but we are in entire agreement with Weinberg and Ginsbourg (1927) that for the moment these organisms should be regarded as varieties of *Cl. botulinum* and referred to as *Cl. botulinum*, Type C, D or E. Their relationship to each other and to the two classical types is very uncertain, and apart from slight differences in the nature of the toxin produced, there seems to be nothing that would justify their elevation to specific rank."

Bengtson (1924) studied the serological reactions of a number of strains of *Cl. botulinum* concerned in the causation of botulism and grouped them as Types A, B and C. All the strains are anaerobes and suitable conditions are necessary for their cultivation in media. After a comparative study of over 100 strains of the bacillus, Meyer (1922) expressed the opinion that growth and toxin production takes place best at 35° to 37° C. One tenth of the amount of oxygen present in the atmosphere will inhibit the growth of all the types (Dack and Baumgartner, 1928). Types

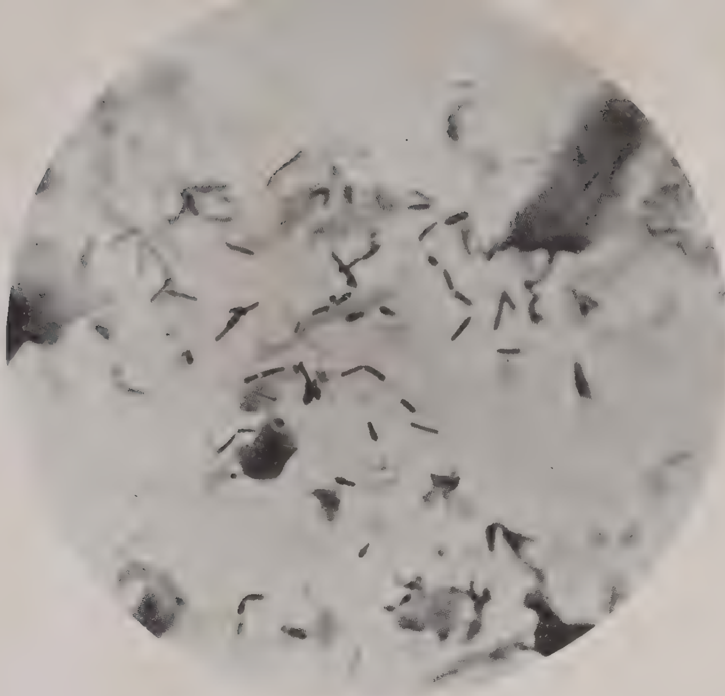

Fig. 37.—Cl. Botulinum, Type A.

Fig. 38. Cl. Botulinum, Type B.

Fig. 39. Cl. Botulinum, Type C.

FIG. 40. DR. J. G. GEIGER.

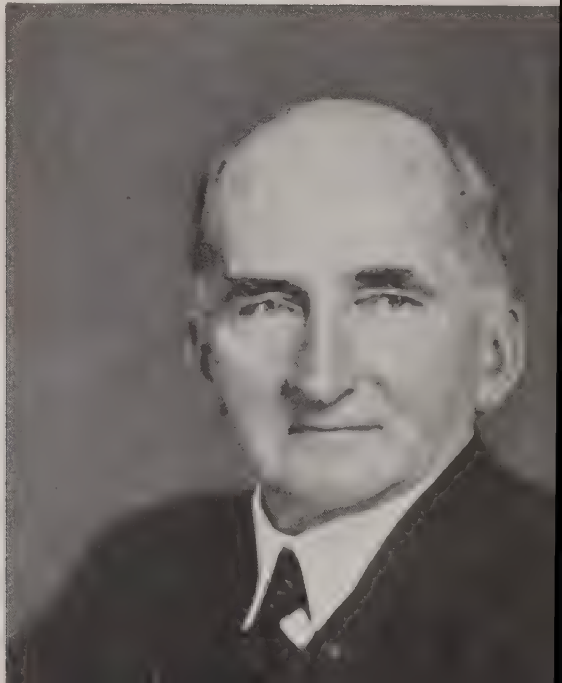

FIG. 41. DR. GERALD R. LEIGHTON.

A and B can grow in approximately 7·5 per cent. of normal atmospheric $O_2$, but Type C will develop only when the amount of oxygen is less than 3 per cent. of the atmospheric $O_2$ (Jordan, 1931).

It has been found that Types A and B can be distinguished by experimental feeding to chickens, the birds being susceptible to type A only, death occurring in from 18 to 24 hours. Suggestions have been made that this test might prove useful for identifying the strains responsible for cases of human botulism.

Type A, which is a large, thick, motile bacillus, with rounded ends, occurring singly or in pairs or chains, differs in some of its characteristics from Type B in that it can resist comparatively low temperatures. Its subterminal spores are very resistant to heat and under favourable circumstances remain dormant for quite long periods before germinating. Type B more closely resembles B. botulinus, described by Van Ermengem, as it is easily killed by boiling. Types A and B are distinguishable by the fact that the toxin of one is not neutralised by the antitoxin of the other.

Type C is a gram-positive anaerobic spore-bearing, slightly motile bacillus. The organism, however, is longer and more slender than A or B strains and occurs singly or in pairs or chains. Damon (1928) remarks : " The organisms belonging to this type differ distinctly in their cultural characters from the other American strains, but produce a symptom complex in animals that is indistinguishable from that produced by the Type A and B organisms."

The table on p. 134 shows the cultural characters differentiating Cl. botulinum types A and B from Type C and parabotulinus of Seddon.

With regard to carbohydrate fermentation, according to Bengtson (1924) A and B strains ferment dextrose, levulose, maltose, glycerol and dextrin, but fail to ferment galactose and inositol. Type C strains, however, ferment galactose and inositol. Salicin is fermented vigorously by most A and B strains. Indol is not produced by any known strain (Norton and Sawyer, 1921).

## The Occurrence and Distribution of Cl. botulinum in Nature

Available evidence shows that Cl. botulinum is found all over the world, its natural habitat being the surface layers of virgin soil ; it is present, too, in cultivated and other soils. The longer the soil has been in cultivation, the less common is the organism.

The spores of the bacillus may gain access to vegetables, fruits and other cultivated produce and be transported by insects and even swallowed by cattle and other animals.

History relates that Van Ermengem (1897) after his discovery of B. botulinus, endeavoured to prove its existence in nature. He examined 52 samples, which included excreta of domestic animals, intestinal contents of fishes and specimens of soil, mud and manure, but without success. In Berlin, Kempner and Pollack (1897) found a bacillus in a pig's fæces. Van Ermengem examined the organism, which resembled the original B. botulinus, but more closely corresponded to the Darmstadt (1904) type of organism. Dickson (1917) examined the contents of the intestines of 250 grain-fed pigs from San Francisco but failed to isolate the organism.

(After Graham and Boughton)

| | Clostridium botulinum, Types A and B. | Clostridium botulinum, Type C or parabotulinus (Seddon). |
|---|---|---|
| Glucose agar . | Gas. | No gas. |
| ,, ,, . | Disc colonies. | Branching colonies. |
| ,, broth . | Even cloudiness. | Flocculent growth. |
| ,, ,, . . | Acid and gas. | Acid. |
| Meat mash . . | Very fine gas bubbles on surface. | Gas bubbles large and along sides of tube. |
| Milk . . . | No change. | Acid. |
| Motility . . | Motile under cover glass. | Non-motile under cover glass. |
| Spores . . . | Resistant to heat. | Non-resistant to heat. |

The majority of the investigations on the occurrence and distribution in nature of Cl. botulinum has been carried out in America. Burke (1919) studied the subject in California and found that Cl. botulinum was widely distributed in nature and was present in garden soils, thus making it possible for fruits, vegetables, etc., to be contaminated by the spores of the organism. Burke concluded that the bacillus existed near human dwellings and was spread by spiders and other insects, but that the organism was not necessarily associated with the fæces of warm-blooded animals.

The question arose as to whether Cl. botulinum was an intestinal saprophyte and consequently occurred in cultivated regions.

or whether it belonged to the ordinary flora of the soil and could increase under natural conditions.

The positive results obtained from the extensive investigations by Meyer and Dubovsky and their colleagues (1922), who examined 1533 soils of all descriptions, manure, vegetables, etc., collected in the United States of America, Canada, Belgium, Denmark, England (many counties), Holland, Switzerland and other countries, indicate that Cl. botulinum is a natural inhabitant of the soil and of widespread distribution. It also shows that the organism is by no means evenly distributed and is commoner in virgin and pasture than in cultivated soil.

Regarding the types of organisms isolated, Type A was found chiefly in virgin soil. In the 335 samples examined 59 showed Type A and 22 Type B. In the 274 specimens of cultivated soil 18 showed Type A and 16 Type B, and in the 51 pasture samples 3 showed Type A and 11 Type B.

In the European soils Type B was predominant. Of the 64 specimens collected from different counties in England, 5 samples showed Type B.

Type C (Graham and Boughton, 1923–4) was found in soils collected from chicken-runs and stables where outbreaks of limberneck or botulism in horses had occurred.

As to the distribution of Types A and B, Topley and Wilson (1936) remark : " Meyer and Dubovsky's results have not as yet received general confirmation ; some of their conclusions may have to be modified (Geiger and Benson, 1923 ; Bachmann and Hayes, 1924), and more work must be carried out before the relationship of the two types to environmental conditions can be definitely determined."

Leighton and Buxton (1928) made investigations into the distribution of Cl. botulinum in Scottish soils, and examined 160 samples from cultivated gardens, ploughed fields, pasture land and uncultivated waste moorland. Positive results were obtained from 4 of the samples ; pasture land 3, ploughed land 1. Two were Type A, 1 Type B and 1 Type A and B.

Cl. botulinum has been occasionally isolated from the excreta of horses, pigs and cattle, which feed on soil produce (Burke, 1919 ; Tanner and Dack, 1922 ; Easton and Meyer, 1924). Meyer (1924) concluded that the evidence secured from an examination of 95 manure specimens strongly indicates that animal excreta contributes relatively little to the pollution of the soil with Cl. botulinum.

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CHAPTER XV

# SPORES OF CL. BOTULINUM

IF the bacillus is cultivated in a suitable medium at the optimum temperature, spores will usually form and germinate, but in unfavourable conditions their formation is delayed or even prevented altogether.

Regarding the function of spores, it may be of interest to mention that in 1931 Jordan wrote: "Physiologically the spore is usually considered as a resting stage, serving to tide the species over a period of dryness, famine or unsuitable temperature, and to preserve alive in a hostile environment a sufficient number of individuals until such time as favourable conditions recur. In this view the spore stage is physiologically analogous to the periods of hibernation or estivation among higher forms of life, and the living matter of the spore may remain dormant for years or even decades."

According to Dickson and others (1925), the spores of Cl. botulinum retain their vitality for long periods if protected from the action of light and air.

## Resistance to Heat

This has been carefully studied experimentally in America by many observers. The destruction of the spores of Cl. botulinum, which can withstand high temperatures for long periods and boiling water for half an hour to 22 hours (Bigelow and Esty, 1920; Weiss, 1921; Esty and Meyer, 1922; Tanner and Twohey, 1926), and 120° C. for 20 minutes, is an important consideration in the prevention of human botulism.

The spores of some strains of Cl. botulinum are more distinctly heat-resistant than those of any other anaerobes. The fact that delayed germination of the spores sometimes takes place, even after they have been subjected to a comparatively high temperature, adds to the difficulty of determining their heat-resistance. Jordan (1931) remarks: "Indeed, if the germination of the spores be inhibited so that growth and consequent toxin production are prevented, botulism cannot occur."

It has been recognised that in canning and preserving foods, acidity (the intensity factor of acidity, not the percentage of acid

present) is one of the chief factors affecting time and temperature for the destruction of the spores of Cl. botulinum. In other words, a close relationship exists between their heat-resistance and the hydrogen-ion concentration which is expressed in terms of *p*H value. Products with a *p*H value below 4·5 are not usually subject to spoilage when packed under satisfactory sanitary conditions. The higher the hydrogen-ion concentration the shorter the time required for the destruction of the spores. The hydrogen-ion concentration necessary, however, to inhibit their development varies according to the nature of the acid and the specific strain Meyer (1928). In the medium in which they are heated, the spores germinate freely at a *p*H value of 6·0 to 7·2.

Esty and Meyer (1922), who carried out extensive investigations, found that at *p*H 7·0 the spores were killed in 330 minutes at 100° C.; at *p*H 5·05 in 45 minutes, and at *p*H 3·7 in 10 minutes. The smaller the number of the spores in the food the shorter is the time necessary to destroy them (Bigelow and Esty (1920)).

Esty (1923) found that the spores of Type A were more resistant than those of Type B. Type C strains form less resistant spores. After much intensive experimental work, Esty came to the conclusion that all the spores of Cl. botulinum would be destroyed at the following times and temperatures:

| | |
|---|---|
| 100° C. | 360 mins. |
| 105° C. | 120 ,, |
| 110° C. | 36 ,, |
| 115° C. | 12 ,, |
| 120° C. | 4 ,, |

NOTE.—Pure water which is neutral (neither acid nor alkaline) has a *p*H value of 7. A large number of foods are more or less acid. The more acid the food, the lower its *p*H value—the more alkaline it is, the higher the *p*H value.

There is considerable variation in the heat-resisting properties of spores of different strains, and even spores of the same strain even under controlled experimental conditions. Tanner (1933) remarks: "The heat-resistance of spores in nature is probably quite different from that of spores under artificial conditions of the laboratory. Practically all our data on heat-resistance have been secured on spores grown in the laboratory. In many cases the menstrum in which the spores were suspended is an unusual one and not like those in food."

The spores of Cl. botulinum are not killed by weak acids or by fairly strong brine concentrations. Food containing salt (sodium

chloride) lowers thermal resistance, which decreases with increasing concentration of the salt (Weiss, 1921).

According to Esty and Meyer (1922), no decrease in their resistance to heat was noticed until 8 per cent. salt solution was reached. Spores are not destroyed by prolonged exposure to cold; they can survive freezing at − 16° C. (− 3·2° F.) for 14 months, as shown by Wallace and Park (1933).

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CHAPTER XVI

# BOTULINUM

## Toxin of Cl. botulinum

THE extremely poisonous substance produced during the growth of Cl. botulinum (Strains A, B and C) in a suitable culture medium under strict anaerobic conditions and at a proper temperature, is a powerful filterable bacterial exotoxin possessing certain physical and chemical properties. Its production is more uniform when glucose is present in the nutrient medium.

The virulence of the poison varies greatly ; it depends upon the strain of the organism, medium temperature and conditions of anaerobiosis (Dickson, 1918). Not all strains form toxin, but the majority do (Bengtson, 1922 ; Starin, 1924).

Meyer (1929) found that an incubation period of 10 days at a temperature of 35° to 37° C. produces a toxin of the highest potency. It may, however, develop in small quantities below 20° C. and up to 34° C.

Dickson (1918) during his investigations and experiments found that the strongest toxin was produced in pork and beef infusion, but virulent poisons were also excreted in media prepared from string beans, green peas, and green corn, respectively. Much less virulent toxins were obtained in media prepared from asparagus, artichokes, peaches, apricots and crushed apricot stones.

The toxin is insoluble in alcohol, ether or chloroform. Cold may act as a deterrent to its development in food, but as soon as the latter (containing the spores) is warmed up, toxin formation may occur.

Wallace and Park (1933) showed that no decrease in potency occurred when the poison was stored at − 79° C. (− 110·2° F. (for 2 months or at − 16° C. (− 3·2° F.) for 14 months.

Under experimental conditions the toxin is destroyed by the prolonged action of direct sunlight, diffuse daylight and air (Schoenholz and Meyer, 1924 ; Bengtson 1924), but if kept sealed and in the dark, it retains its potency for long periods. Putrefaction has no effect on its virulence if access of air is prevented (Dickson, 1918).

It is resistant to acids. Van Ermengem (1896) observed that tartaric and lactic acids in the proportion of 1 to 3 per cent. and

hydrochloric acid in the proportion of 0·5 to 1 per cent. did not lower the toxicity of a filtrate after incubation from 24 to 36 hours at 35° C.

Bronfenbrenner and Schlesinger (1924) found that the poison resisted acidity equal to that of the stomach for 24 hours at 37° C. and noticed that the potency was increased by acidification.

Alkalis exert a powerful effect upon the toxin as observed by Van Ermengem (1896). This was confirmed by Landmann (1904) and by Bronfenbrenner and Schlesinger (1924).

The poisonous property of the toxin is such that the fatal dose for an adult man, calculated on the basis of animal experiments, might be as small as $\frac{1}{100}$ mgm. or even less.

The three types produce toxins of different potency, the ratio of lethal dose for Types A, B and C, respectively, being approximately 1 : 50 : 125 (Bengtson, 1924).

As illustrating its highly poisonous and fatal nature, Dickson (1918) records the case of a woman who died after nibbling a portion of a pod of spoiled string beans and of another who succumbed after tasting a small spoonful of spoiled corn. He remarks : " It is known that the sub-lingual mucosa permits fairly rapid absorption, and it is possible that the toxin may be absorbed in fatal quantities by this route."

The poison after ingestion resists the gastric secretions and is absorbed by the mucosa of the stomach and upper intestine without undergoing alterations and gives rise to the disease.

The action on human beings is obscure, but its principal effect is upon the motor nerve endings. Dickson (1918) states : " It is possible that the toxin acts, as does belladonna, upon the terminal end-plates of certain nerves, and the close resemblance between the effects of the botulinus toxin and those of the administration of belladonna suggest that this may be true."

Minute quantities of the toxin are fatal when introduced into suitable experimental animals (rabbits, monkeys, cats, pigeons, etc.) either by subcutaneous, intraperitonial or intravenous injection, or by feeding. As little as 0·0003 to 0·001 c.c. of a broth culture may kill a rabbit. The poison produces all the characteristic symptoms of botulism (Savage, 1920).

In laboratory experiments it has been possible to obtain a toxin of which 0·000001 c.c. will kill a 250 grm. guinea-pig in from 3 to 4 days (Brieger and Kempner, 1897).

Researches by Geiger (1924) indicate that it is possible to poison experimental animals by absorption of the toxin through

lacerations in the gums or abrasions in the skin or from uninjured mucous surfaces. "Therefore extreme care should always be taken in handling suspected contaminated packs of food, though it is fully recognised and appreciated that from the epidemiology of the majority of the botulism outbreaks that have occurred, this is a remote possibility."

It is true that there is no proved record of the poisoning of human beings except when the toxin is ingested.

Unlike the spores of Cl. botulinum, the toxin is quickly destroyed by heat, but the time taken for destruction varies with the strain of the bacillus and the temperature. According to Jordan (1931), "exposure for from 6 to 10 minutes at 80° C. is sufficient to inactivate the toxin produced by most Type A strains; the Type B toxin needs a somewhat longer exposure (15 minutes), and the Type C toxin is still more resistant (up to 30 minutes at 80° C.)."

## Botulinum Antitoxin

The toxins of the various strains of Cl. botulinum give rise, when suitably injected, to specific antitoxins. The specific antitoxin of one type, however, will not protect against the toxin of another type. This is contrary to the situation observed in the case of other toxicogenic organisms (Damon, 1928).

Jordan (1931) states: "It is a remarkable fact that the characteristic physiological action of the three toxins (A, B and C) seems identical, that no marked cultural difference between the types has yet been made out and that the agglutination reaction shows intergrading of the types as well as differences within each group. The only basis on which a type distinction seems warranted is the specific nature of the antitoxin."

Early experimental work on antitoxin production and its therapeutic properties was carried out by Kempner and Pollack (1897). They also demonstrated the curative value of the serum and found that when the serum was subcutaneously injected into guinea-pigs after the appearance of symptoms of intoxication, some of the animals recovered, in others death did not supervene for weeks or months. Thus a certain prophylactic value for the antitoxin was proved.

The antitoxin sera can be prepared by the injection of goats (Kempner, 1897; Forssman, 1905); horses (Leuchs, 1910); rabbits (Nevin, 1921), with each type of toxin.

Damon (1928) points out that "the curative value of antitoxin

in human cases has not been definitely established, but there is some evidence that it may be employed effectively in prophylactic doses. The curative property depends on the elapsed time since the ingestion of the toxic food and the amount of toxin consumed."

Favourable results in human cases have been reported (McCasky, 1919; Geiger, 1920). According to Topley and Wilson (1936), "large doses, 50 c.c. or more, of polyvalent serum, or of monotypical serum if the type of the intoxicating organism is known, should be given intravenously every day till the patient recovers, or all hope is abandoned. A prophylactic dose of 10 c.c. should be given intramuscularly to all who have partaken of the poisonous food and who have not yet developed symptoms of the disease."

The Ministry of Health has made arrangements for a suitable supply of Botulinus Antitoxic Serum to be available for Medical Officers of Health and Medical men in case of need at several centres in England and Wales.

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CHAPTER XVII

# KINDS OF FOOD ASSOCIATED WITH OUTBREAKS OF BOTULISM

HISTORY relates that the incriminated foods in the majority of the earlier cases of so-called ‘ sausage poisoning ’ were of animal origin, i.e. blood or liver sausage, blood puddings and other ‘ made-up ’ meat foodstuffs. The ingredients used in the preparation of these articles consisted chiefly of liver, sheeps’ brains and plucks, tongues, veal, pork, calf or goats’ blood, and fats of various kinds. They were packed in skins or casings (stomachs or large intestines), which were easily procurable and inexpensive, but on account of their nature and size, difficult to smoke satisfactorily. Being uncooked or only partially so, they did not resist putrefaction to any extent ; moreover, according to historical records, were sold to the poorer classes and sometimes eaten raw. Small meat sausages were also manufactured and apparently prepared and smoked or cooked by skilled workmen under improved sanitary conditions and more expensive.

It is an interesting and significant fact that Kerner (1820) noted that when the sausage casings were incompletely filled they did not become toxic, and he concluded that exclusion of air was necessary for the development of the poison which caused the characteristic symptoms of the illness. Kerner also observed that the poisonous food had a peculiar odour, differing from that of putrefaction.

In recent outbreaks of botulism in Central Europe, other foods of animal origin were implicated. These were in more or less spoiled condition, probably due to being partly smoked—incomplete impregnation with the antiseptic substances of wood-smoke or inadequately home-pickled or insufficiently cooked. Among these foodstuffs were smoked, pickled or salted hams or fish, pork brawn, preserved meats, game *pâtes*, potted or smoked goose or duck, etc.

It has been suggested that in the case of hams the contamination was introduced through the bone marrow. Toxic portions have often been found in the deeper parts, which is favourable for the growth of the spores of B. botulinus. The only German outbreak which was definitely traced to vegetables (string beans) occurred at Darmstadt in 1904.

Geiger, Dickson and Meyer (1922) express the opinion that "the food and food products responsible for botulism in Germany were primarily home-preserved. It is also pointed out that the prevalence of botulism in country districts frequently is due to inadequate preservation on account of the careless and unsanitary treatment to which the raw material is subjected by the rural population. Home slaughtering and preservation of pork products in form of sausages is so universally practised in Germany that it is not at all surprising to find that about one-half of the botulism outbreaks were caused by this type of food. Inasmuch as many of the German records and histories are rather vague and indefinite, it seems, however, hardly fair to draw further comparative deductions."

In America, although meat and preserved meat products, sausages, fish, shell-fish, cheese, etc., were among those foods formerly associated with cases of botulism, the majority of outbreaks in recent years have been attributed to canned or bottled fruits and vegetables. These included apricots, asparagus, string beans, beet, olives, onions, pears, peas, spinach and sweet corn. They were mostly home-preserved in cans or glass jars and consumed cooked or uncooked in the form of salads. "Home-canned string beans alone accounted for 19 out of 55 outbreaks of botulism" (Topley and Wilson, 1936).

Geiger, Dickson and Meyer (1922) record that in 33 outbreaks plant products caused 72·5 per cent. of the cases, while 2·73 per cent. were attributed to animal products. Commercially canned spinach and home and commercially canned string beans were responsible for one-half of the single or group family cases. The wide distribution of Cl. botulinum in the soils of America seems to offer sufficient explanation of the contamination of so large a variety of fruits and vegetables.

Meyer (1936) in recording the foods responsible for 261 outbreaks in the United States and Canada during the years 1899–1935, points out that "although under-sterilised plant products were involved in 76·3 per cent. of the cases, it is important to emphasise that animal products (14·9 per cent.) continue to play a rôle in the outbreaks. Among the newer foods associated in the recent fatal cases, home-canned pork, salmon and crab meat must be mentioned."

Jordan (1931) remarks: "There is, moreover, a significant uniformity in the type of food implicated. Fresh food, raw or cooked, is not the bearer of botulinum toxins. Practically all the

reported cases of botulism have been caused by food that has been given some sort of preliminary treatment, as smoking, pickling or canning, then allowed to stand for a time, and eaten without cooking. Most of the recent outbreaks were due to home-canned vegetables processed in boiling water. Provided the food substance is not too acid or too alkaline and is shut off from free access of air, almost any food seems able to serve as a culture medium for the specific bacillus."

The foods responsible for the cases of botulism which have occurred in Great Britain were as follows :

Loch Maree, Scotland (August 1922). Potted wild duck paste in glass containers (commercially preserved). The preparation of the paste was given as follows : The meat was boned and weighed, and afterwards cooked in an open cooker for an hour or two. It was then transferred to machines to cut it up and reduce it to a paste and the flavouring ingredients added. The mass was placed in large shallow pans, two feet square and three inches deep for sterilisation under 10 lbs. pressure for two hours at 237°–240° F. The meat was removed from the sterilisers and stirred with sterilised paddles. It was then placed in the filling machines and delivered into glasses which were capped and cooked at 210° F. for 40 minutes. Regarding the above process, Tanner (1933) remarks: " In the light of the work which has been done in the United States on the heat-resistance of the spores of Clostridium botulinum, such cooking would not destroy even some of the weaker spores, to say nothing of those which have been shown to be especially resistant to heat. The procedure for the preparation of this duck paste, together with the chemical constitution, make it an ideal product in which Clostridium would form its toxin. If there were a few spores of the organism present in the beginning, they would be disseminated throughout the paste. The slight heating which the paste received might reduce the content of other organisms which could have an antagonistic effect on the toxin producer and thus give the Clostridium botulinum freer range."

North London (August 1935). " The incriminated ' vegetable brawn ' consisted of a mixture of various vegetables (carrot, turnip, peas, beans, vegetable marrow, etc.) with ground-up nuts of various kinds, breadcrumbs, flour, herbs, spice, and hard-boiled eggs embedded in an agar jelly flavoured with marmite. Altogether some 60 jars, each of rather less than a pound, had been manufactured during the month preceding the outbreak ; none of

these except the two responsible for the outbreak was found to contain the specific toxin (12 examined). The vegetables were steamed for about 20 minutes before being added to the brawn mixture. The whole mixture was then placed in glass jars, sealed with airtight lids and steamed at the boiling-point of water for 2 hours, the same process having been used for 30 years. Cl. botulinum was isolated from the remains of the nut-meat brawn consumed by the patients who died of botulism. Its characteristics were those of Type A " (Ann. Rep. C. Med. Officer Min. Hlth., 1935, p. 151).

North London (August 1935). Steak pie. Cl. botulinum, Type B, isolated from the pie. This was the first occasion this type has been obtained in this country in connection with a human case.

## Physical Appearance

The early history of botulism records that the contaminated foods were spoiled or decomposed. In recent years, although this condition has not been observed in every instance, in the majority of outbreaks the foods showed more or less marked changes from the normal or were noticeably spoiled. In the case of home-preserved fruits and vegetables in jars, bubbles of gas were present and the liquid squirted out when the tops were unscrewed. The contents had a disintegrated appearance, a bitter taste and gave off a smell like rancid cheese or butter. When the food was preserved in cans, these were sometimes blown and the contents had a mushy appearance and rancid odour. In occasional instances, however, the canned food presented no abnormality in consistence or odour although the toxin was present. In other cases, the rancid smell was only noticeable when the food was heated, while the physical disintegration was slight. Cloudiness of the brine or liquor may be the only sign of bacterial activity.

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# ILLUSTRATIVE OUTBREAKS

## The Loch Maree Tragedy in Scotland

THE first recorded outbreak of botulism in the British Isles, as before mentioned, occurred at Loch Maree, Gairloch, Rosshire, in August 1922. A party of visitors staying at a local hotel went out fishing on the morning of 14th August and during the day partook of some sandwiches which contained potted wild duck paste.

About 3 a.m. on the 15th one of the visitors was taken ill, and later several of the others complained of illness. The first death occurred at 9 p.m. on the 15th, and in all 8 persons died during the ensuing week.

The following tables from Leighton's work on Botulism give a summary of the symptoms of each case, together with period of onset and duration of illness.

TABLE OF SYMPTOMS

| Patient. | Age. | Dizziness. | Double Vision (Diplopia). | Paralysis of Eyelids (Ptosis). | Paralysis of Speech. | Paralysis of Swallowing. | Respiratory Distress. | Reflexes Diminished. | Cardiac Failure. | Intense Restlessness. | Vomiting. | Pupils Dilated. | Headache. | Some Pain. | Diarrhoea. | Fever. | Loss of Consciousness. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Mr. S. | 70 | × | × | × | × | × | × | × | × | × | × | — | × | — | — | — | — |
| Mr. W. | 66 | × | × | × | × | × | × | × | × | × | × | — | — | — | — | — | — |
| Mrs. D. | 56 | × | × | × | × | × | × | × | × | — | — | — | — | — | — | — | — |
| Mr. D. | 60 | × | × | × | × | × | × | × | × | × | — | — | — | — | — | — | — |
| Mr. T. | 22 | × | × | × | × | × | × | × | × | — | × | × | — | — | — | — | — |
| Mrs. A. | 45 | × | × | × | × | × | × | × | × | × | × | — | × | × | — | — | — |
| K. McL. | 35 | × | × | × | × | × | × | × | × | × | — | × | — | × | × | — | — |
| J. McK. | 40 | × | × | × | × | × | × | × | × | — | — | — | — | — | — | — | — |

PERIOD OF ONSET AND DURATION OF ILLNESS
(Lunch assumed at 1 p.m., 14th August)

| Patient. | Age. | Onset, before Symptoms Appeared. | Duration, after Symptoms Appeared. |
|---|---|---|---|
| Mr. S. . . . . | 70 | 15 hours | 17 hours |
| Mr. W. . . . . | 66 | 14 „ | 21½ „ |
| Mrs. D. . . . . | 56 | 17 „ | 18 „ |
| Mr. D. . . . . | 60 | 17 „ | 6 days |
| Mr. T. . . . . | 22 | 20 „ | 24½ hours |
| Mrs. A. . . . . | 45 | 18 „ | 46 „ |
| K. McL. . . . | 35 | 26 „ | 46 „ |
| J. McK. . . . . | 40 | 44 „ | 5½ days |

It will be seen that the shortest interval between the ingestion of the incriminating food and the onset of the symptoms was about 14 to 18 hours and the longest 44 hours.

Samples of the remains of some of the wild duck paste, as well as that in one of the sandwiches, were bacteriologically examined at the University of Bristol by Bruce White. A long series of cultures were instituted on various media and the organisms grown under both aerobic and anaerobic conditions.

According to Bruce White's report, he says: "Of these cultures, all the sandwich meat cultures and all the wild duck cultures were found to be terribly pathogenic to mice when minute quantities were injected subcutaneously. The cultures giving positive results were now closely scrutinised. Microscopically the wild duck cultures had every appearance of purity, consisting entirely of large bacilli producing egg-shaped terminal spores. An anaerobic sporing bacillus had been isolated from the wild duck paste and was highly pathogenic to mice. As soon as full cultures had been set up experiments were initiated to test the toxicity or otherwise of the samples themselves."

The conclusions which the bacteriologist arrived at were as follows:

"1. The wild duck paste contained a potent toxin, the action of which is inhibited by botulinus (Type A) anti-toxin.

"2. The anaerobic spore-bearing bacillus isolated from the 'wild duck' paste produces a similar toxin which is likewise counteracted by botulinus (Type A) anti-toxin.

"3. The identity of the wild duck bacillus with B. botulinus (Type A) seems established, as also the identity of botulinus (Type A) toxin and that of the wild duck paste.

"4. It seems certain that the wild duck paste and the sandwich were the only toxic foodstuffs submitted for examination."

Further interesting experiments were subsequently carried out upon mice. A very minute quantity of the wild duck paste was made into an emulsion and injected into three mice. All the mice died. Similar injections were made into three other mice, but in addition each was given a dose of anti-toxin along with the poisonous emulsion. These mice remained completely protected by the anti-toxic serum.

The bacteriological examinations and the experiments carried out definitely proved the presence of B. botulinus and its toxin in

the suspected wild duck paste, and that the outbreak was due to the ingestion of the sandwiches containing this paste.

## The United States of America

### Home-canned Asparagus (Dickson, 1918)

On Saturday the 24th November 1917, Mrs. E. of Seattle, Washington, opened a jar of home-canned asparagus and cooked half of the contents. None of the persons who ate the cooked asparagus suffered any ill-effects. On the following evening she ' warmed up ' the remainder of the asparagus from the jar by placing it for a few minutes in warm but not boiling water. Her husband stated that this asparagus did not taste very good, but he ate it all. On Tuesday afternoon Mr. E. complained of disturbance of vision, was nauseated and vomited. On Wednesday morning he was very weak, vomited again after taking food, and had severe diarrhœa. The diarrhœa continued during the day and following night, and during the night the patient complained of cramps in the legs. There was no abdominal pain during this time and no disturbance of sensation. During Wednesday afternoon he began to have difficulty in talking. On Thursday Mr. E. was unable to sit up because of weakness, and he complained that he could not hold up his head. He was unable to speak intelligibly and he complained of dryness in the mouth and pharynx. During the afternoon he began to have difficulty in swallowing and by evening " all the water returned through his nose." He had much difficulty in clearing thick, tenacious mucus from the pharynx and had severe strangling spells when he attempted to swallow. There was no disturbance of mentality, no pain except the cramps in the legs and no fever. He was found dead in bed early Friday morning, about 2 hours after he had succeeded in swallowing a small quantity of milk.

On Thursday, 29th November, another jar of the same lot of asparagus was opened and was served cold as salad at the Thanksgiving dinner. Two persons partook of the salad and both developed symptoms and died. The remnants of the salad was fed to the chickens, and all the chickens developed typical symptoms of limberneck and died. Bacteriological examination was made of the contents of the crops and gizzards of 10 of the chickens and from 6 of them a virulent strain of B. botulinus was isolated.

The asparagus had been purchased in the open market and canned at home by the method described in the pamphlet issued

by the manufacturer of the glass jars which were used, with the exception that it was not parboiled or blanched before it was packed into the jars. The asparagus was washed in cold water, packed into 1-pint and 1-quart jars, which had been boiled, and covered with cold water to completely fill the jars. One half teaspoonful of salt was added to each jar and the covers loosely applied. The jars were immersed to the neck in a wash-boiler, which had a tightly fitted cover, and were allowed to remain for 3 hours after the water began to boil actively. On removal from the boiler, the jars were tightly sealed and placed in a dark closet.

It is interesting to note that in the above outbreak none of the persons who partook of the cooked asparagus suffered any ill-effects, whereas the man who ate the remainder of the asparagus from the jar uncooked developed the typical symptoms of botulism.

**Home-canned Apricots** (Dickson, 1918)

On Sunday the 27th January 1918, a party consisting of 9 persons, 5 adults and 4 children, had supper together near Madera, Calif. The supper consisted of fresh pork, brown beans, bread, butter, milk and home-canned apricots. It was noted that the apricots had a peculiar taste, but 8 of the party ate some of them. The only member of the party who escaped illness was the one who did not eat any of the apricots.

On Tuesday morning, 29th January, 3 of the children, aged 3½, 5 and 14 years, respectively, complained of seeing double, and 3 of the adults complained of dizziness. The 3 adults also developed diplopia during the day. The fourth adult first showed symptoms of illness on Tuesday night, and the smallest child, aged 13 months, became ill on Wednesday evening. One of the children died on Wednesday morning, 30th January, two on Wednesday evening and one on Friday morning. Two of the adults died during the night of Thursday and the remaining two have apparently recovered after a prolonged illness.

The symptoms of all the patients were practically identical except in degree of severity. In all there were dizziness, weakness and inco-ordination of muscular movement, early disturbance of vision with blepharoptosis, mydriasis and diplopia, difficulty in swallowing and talking, and strangling spells induced by attempts to raise thick mucous from the pharynx or to swallow. In one case, one of the patients who recovered, there were initial diarrhœa and vomiting which occurred from 12 to 15 hours before the onset of the eye symptoms, but in none of the other cases were there

acute gastro-intestinal manifestations. In all the cases there was persistent constipation.

On Wednesday, 29th April, the portion of the apricots which remained in the jar was thrown to the chickens. On Thursday several chickens showed signs of limberneck and some of them died, and by Friday afternoon over 25 chickens and 1 turkey had died. A wild canary with similar symptoms was found lying under a tree and it died a few hours later. Bacteriological examination of the contents of the gizzard of one of the chickens revealed the presence of a strain of B. botulinus which produces a virulent toxin when grown in suitable culture mediums.

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## CHAPTER XIX

# PREVENTION AND CONTROL

THE problem concerning the preventive measures against botulism—which stands alone as a type of food poisoning—does not appear on the surface to be a very difficult one at the present time, as, according to statistics, since the Loch Maree outbreak in 1922, up to and including 1940, there have been only 4 deaths (1935) definitely due to botulism in Great Britain. There are, however, certain important general precautions and measures of control which, from time to time, have been evolved as a result of intensive experimental work.

Botulism is endemic in other parts of the world, where large quantities of canned and preserved foodstuffs are produced, both for home consumption and for export, but more especially in those countries where much home canning and preserving are carried out.

In England, Scotland and Wales, owing to the high standard of efficiency maintained in the inspection of all consignments of canned food at the ports of entry, the community enjoys protection against the possibility of the disease from these sources.

In the United States of America botulism formerly offered a serious menace to the canning industry, but through extensive research and experimentation over a long period, to ascertain the conditions of heating necessary for different foodstuffs, to render them safe for consumption, methods were evolved to eliminate the disease. In commercial canneries, pressure cookers employing live steam are in use, and by scientific tests a correct processing time is determined for each important foodstuff. As a result of the use of this special apparatus and by the enforcement of various sanitary precautions, not a single case of botulism has been traced to ' commercially ' canned food for the past 15 years in the United States.[1]

" The observations of the past 10 years leave no doubt that all canning methods, whether commercial or home, should aim at absolute sterility of the product to ensure freedom from Cl. botulinum or Cl. parabotulinum. In case this pre-requisite cannot be

[1] Three cases and 1 death occurred early in 1941, due to the consumption of a commercially prepared mushroom sauce. (Private communication.)

met, acidification with citric acid, or with a mixture of acetic and citric acids, to a $p$H of at least 4·5 with subsequent heating of the product at 100° for a short period, should be practised. Such vegetables as artichokes, chillies, mushroom sauces, etc., can now be well preserved by the procedure of acidification " (Meyer, 1931).

Mention of a few recent publications, issued in the United States on this important subject, may prove useful for reference by firms in Great Britain engaged in, or contemplating, the preservation by heat of foodstuffs in airtight containers :

" Processes for Non-acid Canned Foods in Metal Containers." Bulletin No. 26L (4th edition), Nat. Canners' Assoc. Research Lab., Washington, D.C., 1939.

" Mathematical Solution of Problems on Thermal Processing of Canned Food," by Charles Olin Ball, Research Div. American Can. Co., Maywood, Illinois, 1928.

" Thermal Processes for Canned Marine Products," by O. W. Lang, Univ. of California Publications in Public Health, 1935.

" What Every Canner Should Know." Bulletin No. 89A, Nat. Canners' Assoc., Washington, D.C.

" Home-canning of Fruits, Vegetables and Meats." Bulletin No. 1762, U.S.A. Dept. of Agriculture.

The Chief Medical Officer of the Ministry of Health in his Annual Report (1935) pointed out :

" It is important that firms engaged in this industry should be well-informed on this subject and should be equipped with appliances which are capable of doing the necessary sterilisation and are properly operated to this end. Some firms may not devote the attention which they should to this aspect of their business, and if this is so they constitute a menace to the consumer and to the reputation of the trade as a whole."

## Home-canning and Preservation

The fact that Cl. botulinum occurs naturally in the soil and is widely distributed throughout the world, makes any attempt to avoid initial contamination of fruits and vegetables difficult. The organism thrives and multiplies on decaying vegetation, and whenever spoilage of the raw product occurs, any spores present may rapidly increase in numbers.

The home canner naturally purchases vegetables or fruits in the cheapest market, and these may have been stored for several

days. Poor quality articles, especially vegetables that are heavily contaminated, constitute the principal sources of trouble.

It may be of interest in passing to quote Leighton's (1923) comments on the subject: "It is well known that a large number of ordinary foodstuffs are quite suitable material for the growth of the bacillus in them, and for the formation of its toxin, provided that the special conditions necessary are added. It so happens that these conditions are found nowhere better than in the air-tight container of these preserved foods. If the organism itself is sealed up along with such food without being killed, or if the spores of the organism are so sealed up, having escaped killing on account of an insufficient temperature being applied to the container in the process of sterilisation, then we have all the conditions required for the production of a dangerously toxic product. In such a case the air has been driven out of the container, and with it the free oxygen. The nutrient medium necessary for the growth of the organism is found in the foodstuff itself, and it is only a question of time for the production of the toxin. If the bacillus under these circumstances starts growth there may be production of gas within the container, and other changes which on the container being subsequently opened may be obvious to an observer, and which ought to cause the immediate rejection of the food as spoiled. But it cannot be said that this is always the case, for a number of observers state that in certain cases where the organism has produced its toxin, the preserved food on being opened has shown no obvious change either in smell or appearance."

Generally speaking, in home-canning and preserving the temperature attained in heating is, as a rule, too low to kill any spores of Cl. botulinum.

Fractional distillation, which is sometimes practised, is unreliable, because the spores may not develop in the meantime (Burke, 1919). It is only with steam under pressure that the spores of the bacillus can be destroyed.

A fair amount of home-canning and bottling of fruits and vegetables is carried out in this country, and much useful information and instruction are afforded to individuals attending lectures and demonstrations given by educational bodies. Moreover, useful publications on the subject have been issued from time to time by the Ministry of Agriculture and Fisheries. Bulletin No. 21 (1938) deals with the domestic preservation of fruit and vegetables. This points out that it is more difficult to carry out the canning and bottling of vegetables than fruits.

## Food Poisoning

While the growth of Cl. botulinum and the formation of its toxins are inhibited by acid foods, such as fruits and tomatoes (these can be processed at or near the temperature of boiling water), it is essential for the satisfactory sterilisation of non-acid foods, such as peas, beans and practically all vegetables, that more elaborate equipment should be employed to obtain the high temperatures such as are produced in steam-pressure cookers. These destroy the spores of the organism and prevent any subsequent bacterial growth.

Meyer (1934) remarks : " If there is no pressure cooker available in home-preserving of non-acid foods, it is safer to substitute dehydration, salting or pickling for canning."

## Canning and Preserving Essentials

Here are a few important points and precautionary suggestions in connection with home-canning and preserving :

Vegetables and fruits must be fresh and sound. The former should be young and washed free from dirt and grit and preserved as soon as possible after gathering. If the raw foodstuffs are not required for immediate use, they must be stored under conditions that will prevent deterioration.

Acid-pickled foods require at least 2 per cent. of acetic or citric acid (with a $p$H of 4·0). Brine foods should contain not less than 10 per cent. of common salt.

Vegetables preserved by the ' cold pack ' method must never be served as salads unless they have been previously boiled.

These simple and inexpensive methods of preservation have one safeguard, i.e. the food is not ready to be served from the container but requires soaking in water and sometimes subsequent boiling, which is of course the most effective precaution.

All bottles and cans of preserved food must be carefully examined before opened. There should be no bulging of the tops of the bottles and no escape of gas or liquid when opened. The ends of the cans should be flat or curved slightly inwards ; the inside, smooth and not corroded and the odour characteristic of the product. Never taste food to discover spoilage.

Preserved food which by a rancid or butyric-like odour arouses suspicion or shows the least evidence of deterioration must be excluded from consumption.

Jars, bottles or cans suspected of being unsound must never be disposed of indiscriminately, as they may contaminate the soil or even cause botulism in animals or poultry. Burn or bury all

spoiled food. In handling suspected foodstuffs care should be taken to prevent it coming into contact with cuts and abrasions on the hands, as according to Geiger (1924) the toxin may be absorbed by broken skin areas, mucous surfaces and fresh wounds.

Regarding spoilage, Dickson (1918) states : " It is a point of considerable importance that foodstuffs which are contaminated with the toxin of bacillus botulinus may not appear sufficiently spoiled to ensure their being discarded. The vegetables usually have an unpleasant odour and may show bubbles of gas on the surface, but they are not apt to be discoloured or soft and may even appear to be especially well preserved. It should be thoroughly understood that an extremely virulent toxin may produce but little change in the appearance of the food, and the common practice of tasting canned stuff to see whether it is fit for use should be discouraged. All canned food should be discarded if there is any indication that it is even slightly spoiled (this is even more important with home-canned food), and under no circumstances should it be eaten or even tasted before it has been cooked."

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## APPENDIX I

# CONTAMINATION (AND DECONTAMINATION) OF FOODS BY POISONOUS GASES IN WAR

Before discussing the measures necessary for the protection of food against war gases and how food may become contaminated and decontaminated, mention must be made of the various types of poisonous gases most likely to be used in chemical warfare.

The following is a brief description of each :

### Lung Irritants

Chlorine—a true gas—smells like bleaching powder.

Phosgene (Carbonyl Chloride)—a true gas—smells like musty hay.

**Nose Irritants** (Arsenical smokes)

| | |
|---|---|
| D.M. (Diphenylamine Chlorarsine)<br>D.A. (Diphenyl Chlorarsine)<br>D.C. (Diphenyl Cyanarsine) | These are odourless. |

### Tear Gases

C.A.P. (Chloracetophenone)—dispersed in the form of a smoke—odour like apple blossom.

K.S.K. (Ethyl Iodoacetate)—a liquid—odour like pear drops.

B.B.C. (Bromobenzyl Cyanide)—a liquid—odour like bitter almonds.

### Blister Gases

The most dangerous (persistent) gases in chemical warfare.

Mustard Gas (Dichlorodiethyl Sulphide)—a liquid—slight smell like mustard or garlic.

Lewisite (Chlorovinyl Dichlorarsine)—a liquid—odour like geraniums.

### Blood-poisoning Gas

Arsine (Arseniuretted Hydrogen)—an odourless gas.

Calcium arsenide in powder may be used for the production of arsine. The gas is generated when the powder comes into contact with moisture.

The success of any gas attack depends almost entirely on the weather. Favourable conditions are calm weather, absence of sun, low temperature, fog and mist, absence of rain. Such conditions usually occur in early morning, at dusk, on clear or misty nights and on dull or overcast days. Unfavourable conditions are strong winds, sunny weather, heavy rain.

### Detection of Poisonous Gases

Two main groups of methods are in use—the subjective, i.e. gas detection by the senses (particularly the sense of smell), and the objective, i.e. the use of some external chemical or physical aid.

Of the objective methods mention need only be made of the use of yellow detector paint, which quickly changes to red in the presence of a number of the liquid poison gases. The test, however, has certain drawbacks, in that it is useless when affected by liquids; it is not specific as certain of the liquid 'persistent' tear gases will give the same red colour.

### Protection of Food

The use of airtight containers will protect foodstuffs against all gases. Liquid gases will not penetrate glass, polished metal or glazed earthenware.

## Domestic Measures

Foodstuffs should be placed in tins with tight-fitting lids or in bottles having ground-glass stoppers or screw caps and stored in cupboards rather than in larders. Perishable articles, such as meat, fish, butter, etc., where possible, should be adequately protected by transparent cellulose wrapping material, or sealed cartons, waxed or grease-proof paper, or oil skin, or placed in a refrigerator. Foodstuffs must be protected, and any sort of temporary covering affords some protection.

Doors, windows, air gratings and other openings should be closed and protected immediately a gas alarm is given.

The food container is a cylinder constructed of metal, being similar in shape and size to a regulation dustbin, with a special lid. During normal periods the open top of the container is covered with muslin (to exclude flies, etc.) which is secured in position by a piece of elastic, but on gas-raid warning the muslin is quickly removed and the lid lowered into a water-bath around the container,

which thereby forms a perfect seal against the entrance of poison gases.

Practical tests under gas conditions have been applied to the container and the results proved highly satisfactory in every respect.

Note.—Particulars of the "Bristol Domestic Gas-proof Food Container" were kindly supplied by Mr. F. J. Redstone, Food Decontamination Officer, City and County of Bristol.

## Retail Shops

Displays of foodstuffs in windows or on counters should be avoided, and bulk supplies not broken until necessary, i.e. kept in their original packings. Where possible foodstuffs should be stored in rooms, cupboards, drawers or covered metal bins or in refrigerators. A less effective method is to cover stocks with American cloth dust-sheets, corrugated cardboard, sacking or similar material, in order to exclude dust created by explosions and to guard against splashing by poisonous liquids.

Doors, windows and air gratings should be closed and protected during gas attack.

Foodstuffs must also be adequately protected during transport against gas or splashing by poisonous liquids. If available a closed type of vehicle may be used, failing which the goods should be covered by tarpaulins (oil painted). The outer edges of these must be tightly fastened down to the external sides of the vehicle.

## Warehouses and Stores

These should be rendered as gas-proof as possible and similar methods to those mentioned for retail shops adopted. When ventilation is necessary for articles, such as fresh fruits, etc., these should be screened. Where possible a large shed or room (preferably on the first floor), where means for rapid change of fresh air exists, should be utilised and this used for the aeration of gas-contaminated foods.

## Markets

Markets should not be held in the open during air raids. As protection against gas attacks, foodstuffs in markets should be as far as possible retained in their original wrappers, which are relatively gas-proof. Suitable additional coverings (tarpaulins, etc.) must be available for use in case of emergency, bearing in mind

that the spraying of a persistent gas from aircraft may occur. Large quantities of goods in sacks, etc., which are in the open, can be stacked closely together, so that a proportion is protected by the outer layers. Tarpaulins also may be used to protect stores of food in the open. One should be placed closely over the articles, and above this another arranged over a ridge pole like a tent. This will afford air space between the two coverings.

Gas-proofing of indoor markets is essential. This can be carried out by the usual methods, such as closing and sealing all doors and windows just prior to a raid and the market then closed.

## Farms

Dispersion of farm stock is advisable. Stables, cattle sheds and cow byres may be gas-proofed by sealing all air vents and other openings with paper and glue. Blankets soaked in water or a solution of washing soda may be hung over windows and doors.

Ammonia arising from urine in stables, etc., neutralises to some extent Phosgene and Chlorine gases.

### Contamination of Foodstuffs

Contamination depends upon the nature and properties of the foodstuffs concerned and the type of poisonous gas used.

Foods may be divided into groups, as follows :

1. Watery foods—cabbages, lettuces, potatoes, milk and meat.
2. Fatty foods—milk, butter, fatty meat and fish.
3. Dried foods—flour, legumens, preserved cheese, etc.
4. Hermetically sealed foods—tinned meats, vegetables and fruits, bottled vegetables and fruits, jars containing jams, pastes, etc.
5. Growing crops—wheat, barley, oats and fruits.
6. Livestock—animals and birds.
7. Reservoirs—public and private water supplies, ponds, etc.

Watery foods, by the process known as hydrolysis, split up any absorbed gas and may render it harmless.

Fatty foods have an affinity for certain oily gases and vapours.

Dried foods offer resistance to gases according to the compactness of the goods, e.g. flour, sugar, rice, peas.

Hermetically sealed foods are protected if the containers remain undamaged.

Growing crops are not easily affected and generally tend to cleanse themselves.

Livestock are subject to the same disabilities as humans.

Reservoirs, particularly the larger ones under control, are less vulnerable to a gas attack than is popularly supposed.

The extent of the contamination of foodstuffs depends upon the length of time of exposure to, and the properties of, the particular poisonous gas used.

Non-Persistent Gases, such as 'lung irritants,' become fairly rapidly dissipated by diffusion. They produce little effect upon ordinary foods except when these are subjected to prolonged exposure in confined spaces and the gas is highly concentrated.

The Nose Irritant Gases containing arsenic are dispelled by air currents. They are slightly soluble and D.A. and D.C. slowly become hydrolysed by water. D.M. is unaffected by water. Although not readily absorbed by foodstuffs, there is a distinct danger of their causing arsenical contamination to unprotected foods.

The vapours of the lachrymators are not likely to be absorbed even by foodstuffs containing much moisture, except when exposed to high gas concentrations for long periods. C.A.P. is very slightly soluble. If food is badly contaminated by tear gas the taste is unpalatable.

Mustard Gas and Lewisite penetrate dry provisions quicker than watery foods. They have great affinity for fatty foods. Lewisite is particularly dangerous on account of its arsenical content. The liquid forms of these gases cause more serious contamination ; K.S.K. and B.B.C. will make foods repugnant, while Mustard Gas and Lewisite soak into dry goods, the liquid forming a reservoir for continued vaporisation.

## Decontamination (Cleansing)

Under the Defence Regulations, 1939, any necessary steps may be taken by the appropriate authority or duly authorised person to remove and deal with any chattel (which includes food) either by treatment or destruction which has been contaminated by lethal gas or other noxious substance, in order to prevent danger to life or health being occasioned thereby.

The most drastic treatment is the chemical one of destroying or neutralising war gas, but for foodstuffs this is not altogether practicable ; moreover, the difficulty of decontamination may be increased because a single gas may not be used.

The most effective treatments are the simple ones, such as exposure to air, sunshine or water. By these treatments certain

poisonous war gases can be rendered harmless, especially if they are carried out before penetration occurs.

The contamination of food by war gases in liquid form presents greater difficulty. This does not mean that the whole of the food so affected must be destroyed, as certain articles, such as potatoes and oranges, can be washed and aired, which will render them fit for consumption.

Surface layers of fatty foods, such as bacon, butter, meat, etc., can be satisfactorily removed provided proper care is taken not to spread the contamination.

There is no question of an optimum standard during war-time because of the difficulties in obtaining supplies. It is therefore necessary to try and save as far as possible contaminated foods by prompt action and intelligent appropriate treatment.

The table on pp. 164 and 165 shows the effects of poisonous war gases on foodstuffs and the necessary treatment.

## Water Supplies

Mustard Gas and Lewisite in liquid form and arsenical smokes may cause serious contamination in small reservoirs, and although the physiological properties of the arsenical smokes may be destroyed, arsenic is still present in a toxic form. Running water will be affected for a short period if gas bombs fall very close to a stream.

Mustard Gas is very sparingly soluble in water, in which it sinks, and is slowly hydrolysed to give harmless water-soluble products, hydrolysis being accelerated by heat or agitation. In large reservoirs there is little danger of contamination from poisonous gases.

Phosgene and Diphosgene are readily decomposed by water and danger from poisoning is negligible. Chlorine gas can be removed by charcoal filtration. Animals should not be allowed to drink water suspected of contamination, especially if it has an unpleasant smell.

## Food Animals and Poisonous Gases

It is inevitable that in the event of gas attacks animals destined for food will probably fall victims to war gases.

*Vapour.*—If an animal is known to have been affected by a lung irritant gas, or by blister gas vapours and shows symptoms of bronchial or pulmonary trouble, it should be immediately slaughtered before pneumonia develops. The offal should be

WAR GASES AND FOODSTUFFS

| Group. | Gas. | Effects on Foodstuffs. | | | Treatment. |
|---|---|---|---|---|---|
| | | *Food.* | *Effect.* | *Whether dangerous.* | |
| Lung Irritants. | Chlorine, Phosgene } True gases. | Low concentrations will not appreciably affect foodstuffs, and any temporary effect will soon pass off. | | | Spread out and aerate. |
| | | Flour . . | Becomes sour and slightly unpalatable. May make a poor loaf. | No. | 48 hours' airing. Can then be blended with five parts of undamaged flour and will bake normally. |
| | | Bread . . | Outer layers may become unpalatable. | No. | Cut away outer layers and air remainder. |
| | | Cereals . . | Negligible. | No. | 48 hours' airing. |
| | | Meat and fish | May become slightly discoloured on the surface. | No. | Condition improved by airing and cooking. |
| | | Milk . . | May slightly affect taste. | No. | Bring to boil. |
| | | Eggs . . | None. | No. | |
| | | Cheese, butter, margarine, fats | May bleach slightly on surface. | No. | If bleached, cut away affected part which may be used for cooking. Remainder is edible. |
| | | Fresh fruits . | Almost none. | No. | Air and peel off skin or outer layer. |
| | | Dried fruits . | Slight loss of palatability. | No. | Air and cook. |
| | | Fresh vegetables . | Green vegetables may be slightly bleached. | No. | Air and cook. |
| | | Tea and coffee | May become bitter and unpalatable. | No. | None is effective, but such material might be used for blending. |
| Tear Gases. | K.S.K., B.B.C. } Liquids. C.A.P. Not likely to be used. | Owing to the unpleasant smell of the vapours of these gases, there is danger that foodstuffs which have come into contact with them will be unpalatable, e.g. bitter taste after 12 to 24 hours in high concentration on such common foodstuffs as apples, bacon, butter, bread, cheese, flour, sugar, coffee and tea, and liquids such as beer. Splashing shows as yellow or brown liquid. | | | VAPOUR: Spread out and aerate for 24 hours, or until all trace of smell of vapour is dispelled. Guide is palatability. If there is any doubt, the food material should be submitted for analytical examination. Successful salvage will depend upon the extent to which the gas has penetrated the wrappings. LIQUID: If liquid has contaminated the outside of boxes, sacks or other covering, these should be removed as quickly as possible, and the contents aired. |
| Arsenicals. | Arsenical Smokes: D.A., D.M., D.C. | No obvious effects. These gases are readily dispersed and it is unlikely that they will contaminate foodstuffs unless the foodstuffs have been completely exposed to a high concentration. | | | Aeration is the main treatment, but in ALL CASES the food should be held under suspicion in view of the danger of arsenical contamination. |

| | | | |
|---|---|---|---|
| Arsenical and Blister gas. | Arsine :<br>(1) True gas.<br>(2) Powdered form.<br>(3) Lewisite : (Liquid) also a Blister gas. | ditto<br>Vapour only. | ditto<br>Aerate and hold for examination by analyst, bearing in mind that arsenic poisoning is greater than when arsenical smokes are used. Surface layers of fatty foods can be scraped and samples taken. |
| | | Liquid : splashing shows as heavy brown liquid. | Hose down outer casings, and in suitable instances, e.g. canned and bottled foods, swab or immerse in bleach and water to the consistency of a cream. Surface layers of fatty foods can be cut away to a depth of half an inch or more. Afterwards treat as for vapour and aerate, but hold until further samples have been passed by analyst. |
| Blister gas. | Mustard gas. | Vapour only | No arsenic danger. Procedure similar to that for Lewisite. |

LOW CONCENTRATION OF VAPOUR.

| *Food.* | *Effect.* | *Whether dangerous.* |
|---|---|---|
| Bread, flour, cereals, eggs, lean meat, white fish, fresh and dried fruits, vegetables, tea, coffee, sugar, condiments. | In most cases, no ill-effect noticeable after treatment. In others, there may be slight loss of palatability. | No. |
| Milk, cream, butter, margarine, fats, oils, fatty meats, fatty fish, cheese. | Rapidly absorbs gas and may become unpalatable. | Probably. |

| | |
|---|---|
| Liquid : Splashing shows as heavy brown liquid. | ditto |

SPECIAL NOTE.—In hosing down Protected Foodstuffs it should be remembered that—

(a) The most badly splashed receive IMMEDIATE ATTENTION, and
(b) The OUTSIDE of a case or consignment should be ATTACKED FIRST.

In swabbing : work from the outside to the centre and change swabbing material frequently. Used swabs should be either burnt immediately or placed directly into a bin used ONLY for this purpose, the contents of which should be burnt as soon as is practicable.

condemned, but in the absence of any signs of congestion, etc., the carcass can be passed as fit for human consumption. Where the animal has died through gassing by lung irritant or blister gas vapour, the flesh will be congested and dark in colour.

*Liquid Mustard Gas.*—An animal which dies from mustard gas burns will be unfit to eat. One badly splashed by liquid mustard should be slaughtered immediately. The flesh underlying the lesions must be cut away, the offal condemned and the carcass can then be passed for human consumption.

*Arsenical Gases.*—Animals showing signs of having been gassed by Lewisite or arsine, or have eaten fodder contaminated by the former, must be regarded as unfit for human consumption.

## Food Contamination Building

This should be constructed of impervious material and where possible erected on an elevated site, isolated from other buildings. It must be of sufficient size to allow the contaminated food to be well spread out, and be provided with adequate ventilation, as aeration of the food is the main treatment.

The floor ought to be constructed of concrete and properly drained to a suitable type of gully connected to the main drainage, so as to carry away gas-contaminated liquids when hosing down is in progress. Water supply must be direct from the main, but a tank supply should be available in case the water supply service is put out of action.

*Equipment.*—The interior of the building should be furnished with airing racks, trays, trolley and table—all of metal. The equipment must include metal tubular tripod with movable cross-bar and hooks on which to hang meat for inspection and trimming. Knives, bins for storage, pails for bleach solution, should be provided. Tarpaulins should be available to cover food exposed in the open awaiting decontamination if such is necessary.

The dimensions and particulars of the above building are as follows :

*Construction.*—Steel tubular framing covered with galvanised iron. Size, 60 feet by 18 feet. Height, 8 feet to eaves. Total height, 11 feet.

*Site.*—4 inches concrete on a hardcore base. Concrete raft, 63 feet by 21 feet. Washing down platform, 18 feet by 21 feet.

*Openings.*—Sliding track on hangers for five sliding doors to ensure cross ventilation. The 6-inch space at base of aeration room is protected with wire gauze.

*Accommodation.*—Three rooms, viz., Food aeration room, 40 feet by 18 feet. Food storage room, 20 feet by 18 feet, with Office, 7 feet by 7 feet.

## REFERENCES

Ministry of Food, "Food and its Protection against Poisonous Gas," H.M. Stationery Office, 1941.

Ministry of Home Security, "The Detection and Identification of War Gases," H.M. Stationery Office, 1934.

H. Eastwood (1942), "Food and War Gases."

W. R. Wooldridge (1942), "War Gases and Foodstuffs."

## APPENDIX II

*Ministry of Health* *Memo. 188/Med.*

## FOOD POISONING

*Steps to be taken by Medical Officers of Health (outside London) in suspected food-poisoning cases*

In January, 1921, the Ministry of Health issued a circular (No. 165) to Sanitary Authorities together with a Memorandum (Memo. 39/Foods) dealing with the subject of outbreaks of food poisoning, and this was followed in April, 1924, by a circular (C.L. II) offering the services of the Ministry's Pathological Laboratory in London for the bacteriological examination of material obtained in connection with such outbreaks. By this arrangement and the participation of Medical Officers of Health throughout the country, an opportunity was afforded for the elucidation of points in the causation of food poisoning which were still obscure, and much useful knowledge has resulted as to both the bacterial causes and the paths of infection in food poisoning. The observations and their interpretation have formed part of each Annual Report of the Chief Medical Officer since 1924.

It is hoped that in as many instances as possible Medical Officers of Health will continue to take advantage of these facilities which entail no cost to Local Authorities. If, however, they prefer to make or continue local arrangements for the examination of material from food poisoning, the Medical Officer of Health should furnish the Medical Department of the Ministry with details of the bacteriological tests made and the results obtained, in addition to reporting the general circumstances and extent of the outbreak.

It is particularly desired that information of any death or illness in which food poisoning is suspected should be sent to the Ministry at the earliest possible moment. This is important both because the assistance of the Ministry's Laboratory can be most effective in the early stages of an outbreak and because the Ministry are frequently able to offer useful advice and assistance in the immediate measures to be taken. Moreover, it may happen that other cases are occurring in another district due to food from the same original source and early notification will bring the connection at once to light.

With reference to the desirability of early notification, it may be mentioned that specific duties of sending to the Ministry a copy of any special report which he may make to his Authority, and of reporting any serious outbreak of disease to the Ministry, are im-

posed on the Medical Officer of Health under Article 14 (4) and (5) of the Sanitary Officers Order, 1926.

## Methods of Investigation

Food poisoning is divided into two classes: (1) cases due to contamination of food by poisonous chemicals (e.g. arsenic, antimony, copper, lead, alkaloids, etc.); and (2) cases due to bacterial infection of food, these being by far the most frequent—especially with food of animal origin. Cases, however, in which the result of bacterial infection of food is the production of *notifiable* infectious diseases, e.g. scarlet fever or diphtheria conveyed by milk or ice-cream and enteric fever by similar food or by shell-fish, are excluded from this category. Bacillary dysentery may occasionally be indistinguishable clinically and epidemiologically from bacterial food poisoning and should be investigated on similar lines.

As soon as the Medical Officer of Health has established the probability that a particular food, prepared in his district, is at fault, he should at once make detailed investigation into the conditions of its preparation and should obtain material for bacteriological or chemical examination. He will naturally take steps without delay to prevent further consumption of the suspected food by stopping its sale and recovering unconsumed portions already sold.

It will usually be advisable to secure samples from all available food materials in addition to those suspected at first sight, since it sometimes happens that food not originally suspected ultimately proves to be the material at fault. This is of special importance when it is suspected that the illness is due to an inorganic poison.

To confirm the suspicion that a particular food is at fault, a full list of everything consumed at the suspected meal by all the persons present, together with the clinical history of each person attacked, should be obtained as early as possible. The determination of the circumstances in which food poisoning has occurred often turns upon apparently trivial points, accurate recollection of which may be impossible after some days' interval. For convenience of reference a list of headings for inquiry is appended to this Memorandum (Appendix A).

It is not necessary nor is it desirable to await the result of bacteriological or chemical examinations before commencing inquiries as to the manner in which the poisonous elements (bacterial or other) gained access to the food, as supplementary inquiries can always be made when the laboratory findings are known. For

example, if there is any possibility that the food has been contaminated by arsenical or other poisonous substances during transport, inquiries should be made from the railway companies or other transport agencies concerned.

When the food suspected to have caused poisoning has not been prepared in the district, the Medical Officer of Health should gain the co-operation of the vendor who should be invited to produce original packages and invoices, and any facts available to show by what manufacturer or distributor the implicated food was supplied to him, by what route, on what date and in what bulk. The Ministry would be glad to be informed at once of the facts obtained in any such cases.

## Collection of Material

(1) It is important to secure samples of any remaining portions of the *food actually consumed* by persons attacked ; even minute fragments in discarded containers may be of value. Should this be impossible, food of similar origin or prepared from the same ingredients should be collected, though such specimens are much less likely to throw light on the cause. In the case of canned or potted foods the containers with labels intact should be preserved. The experience of recent years suggests that, though almost any food may produce food poisoning, if it happens to have been infected with a salmonella, e.g. by fouling by rats or mice, yet the foods most to be suspected are 'made-up' dishes containing meat, especially pig products. A history of the consumption of ducks' eggs within a reasonable time before onset of illness would suggest attempts to trace the flock from which the eggs came, to obtain eggs from this flock and to examine the blood of the suspected ducks for evidence of recent salmonella infection.

(2) Pathological material should be obtained from the sufferers in the acute stages of the illness whenever possible. Fæces or, failing these, rectal swabs are of the greatest importance ; urine is less likely to give positive results in bacterial food poisoning but it is important when chemical investigation is indicated. Vomited matter is not often of value bacteriologically but should be sent when available. From *fatal cases*, portions of the small and large intestine, spleen, liver, and kidney should be obtained. The stomach (unopened and ligatured with its contents intact) is valuable if metallic poisoning is suspected but not of much use otherwise.

(3) Samples of blood for serological tests (at least 1 ml.) should not be collected until a week has elapsed from the onset of

illness since the agglutinins to be investigated will not have fully developed till then.

## Packing and Transmission

Food specimens and all pathological material should be kept in an ice-box or refrigerator, if delay in dispatch is unavoidable. Specimens of excreta for bacteriological examination should be small in amount ; in the case of fæces containing much mucus, a throat swab dipped in the mucus makes a satisfactory specimen ; otherwise, clean, wide-mouthed, firmly corked or stoppered bottles make suitable receptacles. Food may be put in a clean tobacco or sweet tin. The organs from fatal cases should be wrapped in a clean cloth which has been wrung out of 30 per cent. glycerine solution. Contact with disinfectants must, of course, be avoided in the collection and transmission of all specimens.

It is usually difficult to provide cold storage during transport. It may be improvised by packing the specimens in their containers in a large biscuit tin containing crushed ice and itself packed in a wooden box with at least 2 inches of sawdust or absorbent cotton between. The specimens in their containers should be well wedged to prevent shifting as the ice melts. If ice cannot be easily procured, the specimens should be sent without it rather than be delayed.

The package should be marked ' URGENT ' and addressed to—

Medical Department (Med.I),
Ministry of Health,
Whitehall,
London, S.W.1,

and should be sent by post [1] or by passenger train if more prompt delivery can be thus effected, notifying the Medical Department, as above, in advance if possible.

[1] The following are the current Post Office Regulations regarding articles sent for Medical Examination or Analysis :—

> Deleterious liquids or substances, though otherwise prohibited from transmission by post, may be sent for medical examination or analysis to a recognised Medical Laboratory or Institute, whether or not belonging to a Public Health Authority, or to a qualified Medical Practitioner or Veterinary Surgeon, within the United Kingdom by Letter Post, and on no account by Parcel Post, under the following conditions :—
>
> > Any such liquid or substance must be enclosed in a receptacle, hermetically sealed or otherwise securely closed, which receptacle

## Examination of Specimens

*Chemical Examination.*—When the circumstances point to poisoning not of bacterial origin, samples with all the information available should forthwith be sent for chemical analysis, ordinarily to the public analyst of the area. Little is to be gained by sending specimens of meat foods to the analyst to be examined for 'ptomaines.' It is doubted if 'ptomaines,' in the sense of alkaloidal substances produced by bacterial action in meat foods have any significance or connection with food poisoning. Specifically infected meat foods may, however, require chemical analysis for the determination of special points such as the presence or absence of preservatives and their nature, the determination of acidity, or saltness, and like matters.

*Bacteriological Examination.*—It is of obvious advantage in the investigation of cases of food poisoning that arrangements for any necessary bacteriological examination should have been considered beforehand so as to avoid the delay and trouble of making special emergency arrangements. The exact material required may vary in individual outbreaks, and in all cases the bacteriologist entrusted with the examination should be consulted as to the specimens which will be most instructive.

In Appendix B some technical hints on the isolation and identification of Salmonella types are set out for the assistance of Public Health bacteriologists having to deal with food poisoning material.

It is important that material should be available for any investigations which the Ministry may desire to make through their

must itself be placed in a strong wooden, leather, or metal case in such a way that it cannot shift about, and with a sufficient quantity of some absorbent material (such as sawdust or cotton wool) so packed about the receptacle as absolutely to prevent any possible leakage from the package in the event of damage to the receptacle. The packet so made up must be conspicuously marked " Fragile with care," and bear the words " Pathological Specimen."

Any packet of the kind found in the Parcel Post, or found in the Letter Post, not packed and marked as directed, will be at once stopped and destroyed with all its wrappings and enclosures. Further, any person who sends by post a deleterious liquid or substance for medical examination or analysis otherwise than as provided by these regulations is liable to prosecution.

If receptacles are supplied by a Laboratory or Institute they should be submitted to the Postal Services Department, General Post Office, London, E.C. 1, in order to ascertain whether they are regarded as complying with the regulations.

own officers. Where such an investigation is directed, an early intimation will be sent to the Medical Officer of Health. In all cases, however, it is desirable that the chemist or bacteriologist consulted should be asked to preserve samples under suitable conditions until it has been ascertained that there is no further use for them.

## APPENDIX A

### Headings of Inquiry into Outbreaks of Poisoning by Meat Foods

What cases heard of; steps taken to secure complete list of cases, e.g. inquiries of medical practitioners, neighbouring Medical Officers of Health and others.

Evidence implicating particular food or foods as cause of outbreak.

Evidence implicating any particular ingredient of the food.

Origin of suspected food or ingredient.

### Inquiries in Affected Households

(*a*) Names and ages of persons in each household, (*b*) those ill, (*c*) those partaking of suspected food.

Persons affected (*a*) slightly, (*b*) seriously, (*c*) fatally; with date and hour of partaking of food in each case and date and hour of first symptoms in each case.

Clinical character of illness.

Particular food implicated. Date of purchase, source; any form of domestic preparation applied (e.g. cooking); if so, how long and to what degree; if canned meat, when opened, etc.

### Inquiries at Place of Preparation (when food implicated has been prepared in the District)

Address or description of place of preparation; name and address of owner or occupier; number of workers employed (male and female); nature of employment in each case. Any engaged also in other employments which might have connection with contamination of the suspected food.

Meat concerned; its nature, where obtained, price paid, amount used on days concerned, how and where stored.

Evidence, positive or negative, of unsoundness of the meat.

Evidence, positive or negative, as to disease of animal from which material (meat or milk) was derived during life or ascertained post-mortem.

### Food Poisoning

Possibilities of infection at slaughter-house or place of preparation or storage. Infected rats and mice and the use of bacterial virus as rat poison.

Sanitary condition of bakehouse or food preparing place (including distance from possible sources of contamination, e.g. middenstead, ash-pit, privy, W.C., slaughter-house, stable) ; position of drain openings ; ventilation ; general sanitary condition.

Cleanliness of tables, floors, vessels, utensils, etc.

Preparation of food (exact details of methods employed, including history and condition of various component parts besides the meat, e.g. pastry, stock, and jelly for pork pies, skins of sausages, etc.).

Handling of the food (possible contamination by 'carrier' of bacteria associated with food poisoning (*a*) before cooking, (*b*) during cooking, (*c*) after cooking, e.g. transfer into moulds, etc.).

Temperature reached in cooking ; any experimental verification of temperature especially as regards interior of mass ; any reason to suspect under-cooking of whole or part.

Cooling. Where food placed during cooling. Possible opportunities of contamination.

Health of workers previous to outbreak, especially in regard to diarrhœa ; their habits as to cleanliness. What W.C. accommodation for workers (where situated and condition). Arrangements for washing hands and their use.

### Collection and Examination of Materials for Bacteriological Examination

Samples collected (dates, description and quantities) of—

Food materials : (*a*) Portions left over by patients, (*b*) obtained at shops, stores or places of preparation.

Clinical materials : (*a*) Blood from patients or suspected "carriers," (*b*) post-mortem specimens.

## Headings of Inquiry into Outbreaks of Poisoning suspected to be Due to Food Contaminated with Inorganic Poisons

What cases heard of ; steps to secure complete lists of cases, e.g. inquiries of medical practitioners, neighbouring Medical Officers of Health and others.

Evidence implicating particular food or foods as cause of outbreak.

Evidence implicating any particular ingredient of the food.

Origin of the suspected food or ingredient.

Mode in which the food or ingredient became contaminated.

## Inquiries in Affected Households

(*a*) Names and ages of persons in each household, (*b*) those ill, (*c*) those partaking of suspected food.

Persons affected (*a*) slightly, (*b*) seriously, (*c*) fatally ; with date and hour of partaking of food in each case, and date and hour of first symptoms in each case.

Clinical character of illness.

Particular food implicated. Date of puchase ; source. (The suspected food will usually be some food consumed in common by the persons affected.)

How the suspected food was prepared, and what ingredients were utilised in preparing it.

All suspected food should be secured for chemical examination. If there is any doubt as to the food implicated, all foods taken at the suspected meal and all food materials from which the foods were prepared should be carefully preserved for examination.

## Source of Contamination of the Food

(1) *In the Household.*—Cooking utensils, rat poisons, drugs, etc.

(2) *On Retailers' Premises.*—Storage in proximity to poisonous articles. Examine remainder of consignment from which suspected article was supplied. Take samples for chemical examination. Examine packages or bags for evidence of staining by poisonous material, and take samples of suspicious stains for examination. Trace empty packages or bags from which the suspected food has been sold. Ascertain date of receipt of consignment, date of dispatch from wholesaler and amount received ; also amount sold, dates on which portions were sold and names of purchasers.

Ascertain whence retailer obtained his supplies, the name of the firm supplying him and where the article was prepared or manufactured.

(3) *At Wholesalers' and Manufacturers' Premises.*—Method of preparation of food, opportunities for contamination, ingredients used, their origin, mode of manufacture and purity. Methods if any for controlling purity of supplies.

(4) *In Transit.*—Ascertain modes of conveyance from wholesaler to retailer and from retailer to consumer. Possibilities of contamination in delivery from retailer to consumer, e.g. in vans, carts, etc., containing poisonous substances.

Possibilities of contamination in transit by rail, lorry or barge from wholesaler to retailer. Ascertain whether poisonous articles were packed in the same vans with suspected food.

(It may be remarked that foods contaminated in the course of preparation or by the use of impure ingredients will usually contain the poisonous material, e.g. arsenic, fairly uniformly distributed throughout the food. In the case of foods contaminated in course of transit the poisonous material is commonly not uniformly distributed, e.g. a sack of sugar contaminated by a leaking can of arsenical weed killer may contain only a few lumps of sugar saturated with arsenic ; the bulk of the sugar may remain unaffected and free from arsenic.)

## Specimens for Chemical Examination

In addition to the food and food materials mentioned above, specimens of vomit should be secured, and if a death occurs the stomach and stomach's contents together with a portion of the liver should be reserved for examination.

# APPENDIX B

## The Identification of Salmonella Types

The following technical hints on the isolation and identification of Salmonella types may be of service to bacteriologists doing food-poisoning work.

The routine procedure, on obtaining specimens of suspected food and drink and of fæces and vomited matter from sufferers, is to make direct cultures on plates of MacConkey's lactose bile-salt agar ; appropriate amounts of emulsions of the material in sterile broth or saline, so as to give isolated colonies, are spread *secundum artem ;* the emulsions should be made from various portions of the materials ; mucus, if any is present in the fæces, may be spread directly on the plates, with or without preliminary washing in sterile broth or saline. In addition, a routine 'differential' culture is put up from all the emulsions, etc., in peptone-water (1 per cent. bacto- or other peptone) containing 1 in 150,000 brilliant-green. It is best to use large volumes of this medium, 25 ml. or 50 ml. in appropriate tubes or flasks and to make correspondingly large inocula of the suspected material, up to 0·5 gram. A convenient way is to keep flasks or bottles containing 150 ml. of peptone water and to add, when required, 0·1 ml. of an accurately prepared stock watery solution of 1 per cent. strength of brilliant-green ; this stock solution keeps well at room temperature in the

dark. [Plating on Wilson-Blair bismuth-iron-sulphite-agar (with brilliant-green) is worthy of trial in view of its remarkable efficiency in detecting the typhoid-paratyphoid species, but the Ministry's Laboratory has little experience of its use in food poisoning investigations ; the only disadvantage it might have is an extra day's delay before the appearance of typical colonies.]—After 18–24 hours' incubation at 37° C., the direct MacConkey plates are examined. At the same time the brilliant-green peptone water cultures are plated out on well-dried MacConkey agar ; in doing this, a small loopful (1 to 2 mm. diameter) is taken from the surface film of the culture fluid and allowed to dry on the plate before spreading with a right-angled glass rod. These plates are then incubated till next day at 37° C.

The direct plates, or if these are negative, the plates from the differential brilliant-green peptone water, are examined on a dissecting microscope with a × 6 lens and obliquely transmitted light. It is difficult to express in words the peculiar appearance of Salmonella colonies, but a little experience should enable the observer to pick out, even on a plate crowded with colonies of lactose-fermenting coli and non- or late-lactose-fermenting pyocyaneus-fluorescens-proteus species, the characteristic, pale, finely-structured Salmonella colonies. (In the case of plates from fæces, search should be made also for colonies of dysentery bacilli. These are equally characteristic, being small, flat, bluish colonies with almost no structure in the case of the Flexner species, larger with a tendency to feathery edge with the Sonne species and intermediate with the Newcastle species. In Flexner infections, especially after the acute stage is past, the detection of the dysentery colonies may not be easy ; they often appear as mere sectors of a coli colony which must be picked off and replated.)

The suspected Salmonella colonies, as they are detected on the plate, should be tested forthwith for agglutination. This may easily be done by emulsifying with a straight platinum needle a minute portion of the colony, picked off under the lens, in a drop (loopful) of a group-phase Salmonella serum, such as that prepared by inoculation of a rabbit with ' European suipestifer ' (*Salmonella cholerae suis*, Salmon and Smith) or ' Binns ' (*Salmonella typhi murium*, Loeffler var. Binns), and in a drop (loopful) of a Gaertner serum similarly prepared by inoculation of a rabbit with *S. enteritidis* (Gaertner). A convenient way is to put out on an ordinary glass slide two parallel rows of say 5 or 6 drops, one row of the group-phase serum, the other of the Gaertner serum. The serum in each case should be diluted 1 in 50, or 1 in 100, or 1 in 500 with

saline, the dilution depending on the water-bath titre of the serum ; 1 in 50 will usually be found suitable for sera of 1 in 5000 titre. These dilutions can be kept in small stock bottles, well-corked (not rubber-stoppers) and containing a drop or two of chloroform ; stored in the ice-chest or refrigerator they remain for 10 or more years without much loss of agglutinin.

Five or six colonies are emulsified, each in a pair of drops : it is usually unnecessary to take separate specks of the colony for the two different drops, the amount of serum carried over not being so great as to confuse the reaction. Agglutination appears as a characteristic flocculation which is not readily mistaken for non-specific saline clumping and in any case is distinguishable from this by occurring in one drop and not in the other.

The commonest finding is that some colonies agglutinate characteristically in one or other of the two drops, while other colonies show no agglutination with both drops (or traces only). In the former case, it may be assumed that the colony either is one of a Salmonella in the group phase or is a Salmonella of the Gaertner type (or one of its close relatives) according to which drop shows the reaction. The colonies which fail to react or show traces only either are not Salmonellas or are in the specific phase. In the latter case they will react with their own type serum and the next procedure is to demonstrate this. For this purpose ' pure ' type-specific sera are necessary and the test can be made in drops of dilutions of these in the same manner. These ' pure ' types specific sera may be prepared by inoculation of rabbits with culture entirely in the specific phase, as was originally pointed out by Sir F. Andrewes. But such sera are not always sufficiently free from group agglutinin to give sharp specific reactions ; if they display too much group action, a preliminary absorption of agglutinin with group phase Salmonellas, e.g. absorption of Aertrycke serum with a mixture of Paratyphosus B and Suipestifer, will remove this and leave a serum-dilution giving sharply specific clumping. Such absorbed sera have the additional advantage of containing no ' O ' or ' somatic ' agglutinin which, if abundant in the unabsorbed serum, may blur the sharpness of differentiation. A routine set of such type-specific sera should consist of Paratyphoid B, Aertrycke, Newport, Stanley or Typhosus, Thompson, Morbificans bovis, American Suipestifer and London. Flocculation of a suspected specific phase colony in the characteristic manner with a single one of these then identifies the type with sufficient probability for epidemiological purposes. The type having been thus identified in culture from, say, a specimen of

fæces, search for the same type in the cultures from the food specimens and from other fæcal samples becomes simple; parallel rows of drops of a 'group' serum and the specific serum of the particular type can be made and colonies tested from each MacConkey plate. Some of these will naturally be in the group phase and give only feeble agglutination with their type-specific drop; others, in the specific phase, will clump strongly in the type-specific drop and practically not at all (unless there is much 'O' agglutinin) in the group drop. In rare instances a double Salmonella infection may be present and will not be missed if a reasonable number of colonies are tested in the manner described.

Not seldom, however, the search for 'specific' colonies in plates from the primary material fails; this may be due (*a*) to the temporary suppression of the specific phase, in which case repeated subculture of a group-reacting colony and examination of plates from such subcultures will usually furnish the specific phase, or (*b*) to the presence of Salmonellas such as European Suipestifer [*S. cholerae suis.* (Salmon and Smith)] which are permanently 'group'. In the latter particular instance (European Suipestifer) the cultural characters (absence of dulcitol fermentation and presence of $H_2S$ production) are helpful as fairly constant features of this bacterium. But it may require considerable patience to satisfy oneself that no specific phase exists. Phase-dissociation can often be speeded-up by subculture in broth containing about 10 per cent. of a group serum (e.g. European Suipestifer). An overnight culture in such serum-broth of a group colony will show a dense deposit of bacterial growth with clear supernatant medium; subculture from the supernatant fluid to a fresh tube of the same serum-broth may give, next day a supernatant fluid definitely turbid in which case, when plated out, colonies of the specific phase will usually predominate. In the case of the Thompson type spontaneous phase-dissociation is peculiarly slow and rare; the type appears either as 'pure group' or 'pure specific' in spite of repeated subculture and several passages (up to 10) in group-serum broth may be necessary in order to get colonies in the specific phase.

In the case of the monophasic types which possess a flagellar antigen in common with *S. enteritidis* (Gaertner), of which there are several known and perhaps some still to be discovered, differentiation by means of agglutination in drops of serum is also possible; the type serum in each case (Gaertner, Dublin, Moscow, Derby, Senftenberg-Newcastle) must be absorbed so as to leave the agglutinin characteristic of the type, i.e. the Gaertner serum

must be absorbed with Moscow, the Moscow, Derby, Dublin and Senftenberg-Newcastle sera with Gaertner. With these sera, if of high titre (50,000), a large amount of culture should be used in preparing the stock of absorbed serum and the growth scraped from 2 agar plates (9 mm. diameter) and emulsified in 10 ml. of 1 in 50 dilution of the serum would not be too much. Tests for subsequent type-specificity in drop should, of course, be made before adopting the ' purified ' serum for use.

Rapid identification in drops as above described has been in use in the Ministry's laboratory for ten years. Its practical value is twofold. In the first place, it increases the certainty with which a report can be furnished without delay that food poisoning of the Salmonella variety has occurred and, secondly, it establishes the certainty of suspicions that a particular outbreak is an epidemic unit, or, on the other hand, that cases occurring about the same time are not related to each other, since the infecting type is different.

It is advisable in every case to test one or more colonies culturally as a confirmation of the generic identity (assuming the Salmonellas to constitute a ' genus '). A simple routine set of tests is (*a*) growth in broth (absence of indole) and (*b*) in peptone water containing ' sugars ' as follows : dextrose, mannitol, and dulcitol should be fermented in 20 to 36 hours with the production of acid and usually (but not always) of gas, while lactose and sucrose should not be attacked.

If any doubt still exists as to the serological types, an absorption test, in which the suspected strain removes from the stock type serum all the agglutinins for the homologues, provides confirmatory and practically conclusive evidence. For this purpose titration of the serum, before and after absorption, to end-point in the water-bath at 50° C. is necessary ; drop testing is unsatisfactory for such an absorption experiment.

For complete identification of an atypical strain of Salmonella a much more elaborate piece of serological work is, of course, required, including the preparation of a homologous antiserum in a rabbit, the performance of ' mirror-absorptions,' and the identification of the ' somatic ' or ' O ' antigens as well as the flagellar. But for epidemiological field work the rapid method outlined above will be found useful and reliable ; it is all that is necessary in the majority of outbreaks.

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